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1885 BENNETT, A. HUGHES, M.D., 76, Wimpole Street, W. *Trans.* 2.
1878 BENNETT, STORER, 17, George Street, Hanover Square, W. *C.S.* 2.
1874 BENNETT, WILLIAM HENRY, 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895.) *Trans.* 4, *C.S.* 3.
1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.
1882 BERRY, FREDERICK HAYCRAFT, M.B., Watford, Herts.
1885 BERRY, JAMES, 60, Welbeck Street, W.
1889 BIDWELL, LEONARD ARTHUR, 59, Wimpole Street, W. *Trans.* 1.
1890 BINDLEY, ROBERT ALFRED, Westbury House, Harlesden, N.W.
1882 BINDLEY, PHILIP HENRY, M.B., Branksome Road, St. Leonards-on-Sea.
1879 BINDON, WM. JOHN VEREKER, M.D.
1883 BISS, CECIL YATES, M.D., 135, Harley Street, W. *Trans.* 1.
1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.B., B.S., Belle Vue, Mount Pleasant, Tunbridge Wells.
1892 BLAXALL, FRANK RICHARDSON, M.D., Rendlesham, Hornsey Lane, N.

ELECTED

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.S.* 1.
1888 BOSTOCK, ROBERT ASHTON, 73, Onslow Gardens, Brompton.
1883 BOWLBY, ANTHONY A., 24, Manchester Square, W. (C. 1891–3.) *Trans.* 8, *C.S.* 8.
1883 Bowles, ROBERT LEAMON, M.D., 16, Upper Brook Street, W. (C. 1890–1.)
1889 BOYD, STANLEY, M.B., 134, Harley Street, W. *Trans.* 1, *C.S.* 4.
1890 BRADFORD, JOHN ROSE, M.D., 52, Upper Berkeley Street, W. *Trans.* 1.
1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.
1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.
1868 Bright, JOHN MEABURN, M.D., Forest Hill, S.E.
O.M. BROADBENT, Sir WILLIAM HENRY, Bart., M.D., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.
1887 BROCK, J. H. E., M.D., B.S., 115, Adelaide Road, N.W.
O.M. BRODHURST, BERNARD EDWARD, 20, Grosvenor Street, W. *Trans.* 2, *C.S.* 1.
1894 BRODIE, C. GORDON, 30, Harley Street, W.
1890 BROWN, WALTER HENRY, 19, Queen Street, Leeds. *Trans.* 2.
1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.
1893 BROWNE, JAMES WILLIAM, M.D., 7, Norland Place, Holland Park, W. *Trans.* 1.
1887 BROWNE, OSWALD AUCHINLECK, M.A., M.B., 43, Bedford Square, W.C.
1883 BRUCE, JOHN MITCHELL, M.D., 70, Harley Street, W.
1893 BRYANT, JOHN HENRY, M.D., Guy's Hospital, S.E.
O.M. BRYANT, THOMAS, 65, Grosvenor Street, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 8.
1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., 6, Lower Sloane Street, S.W. *Trans.* 1.
1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 46, Weymouth Street, W.
1881 BURNET, ROBERT WILLIAM, M.D., 6, Upper Wimpole Street, W. *Trans.* 1.
1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.
1887 BUTLER-SMYTHE, ALBERT CHARLES, 76, Brook Street, W. *Trans.* 1.
1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9.) *Trans.* 7, *C.S.* 1.
1871 BUTT, WILLIAM F.
1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.
O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895.) *Trans.* 14, *C.S.* 1.
1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.
1886 CAHILL, JOHN, 12, Seville Street, Lowndes Square, S.W.
1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W.
1890 CALVERT, JAMES, M.D., 36, Queen Anne Street, W. *Trans.* 1 *C.S.* 2.

ELECTED

1893 CAMPBELL, HARRY, M.D., 20, Devonshire Street, W.
1891 CANNEY, H. E. LEIGH, M.B., 122, Brompton Road, S.W.
1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W.
1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. *Trans.* 1, *C.S.* 2.
1889 CARTE, WILLIAM ALEXANDER, M.D., M.Ch., Surgeon-Major, Grenadier Guards' Hospital, Rochester Row, S.W.
1869 CARTER, ROBERT BRUDENELL, 31, Harley Street, W. (C. 1873–6, V.P. 1879–81.) *Trans.* 8.
1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 2.
1868 CAVAFY, JOHN, M.D., 2, Upper Berkeley Street, W. (C. 1881–3.) *Trans.* 4.
O.M. CAYLEY, WILLIAM, M.D., 27, Wimpole Street, W. (C. 1874–5, S. 1876–8, C. 1879–80, V.P. 1885–6.) *Trans.* 8, *C.S.* 2.
1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W.
1884 CHAPMAN, PAUL M., M.D., 1, St. John Street, Hereford. *C.S.* 1.
1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.
1885 CHEYNE, W. WATSON, M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–5.) *Trans.* 1, *C.S.* 5.
1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.
O.M. CHURCH, WILLIAM SELBY, M.D., 130, Harley Street, W. (C. 1874–6.)
1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 5.
O.M. CLAPTON, EDWARD, M.D., 22, St. Thomas's Street, Southwark, S.E. (C. 1872–4.) *Trans.* 1.
1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.
1887 CLARK, FRANCIS WILLIAM, The Dispensary, Newcastle-on-Tyne.
1885 Clarke, J. MICHELL, M.B., 28, Pembroke Road, Clifton, Bristol.
1885 CLARKE, WILLIAM BRUCE, M.B., 46, Harley Street, W. (C. 1894–5.) *Trans.* 2, *C.S.* 1.
1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.
1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *C.S.* 1.
1877 CLUTTON, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7.) *Trans.* 11, *C.S.* 2.
1893 COLE, ROBERT HENRY, M.B., Moorcroft, Hillingdon, Middlesex.
1882 COLLIER, HERBERT, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.
1895 COLLUM, ARCHIE T., M.B., 7, Grosvenor Street, W.
1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.
1872 COOKE, THOMAS, 40, Brunswick Square, W.C.
1868 COOPER, FRANK W.
1880 COTTLE, WYNDHAM, M.D., 3, Savile Row, W.
1892 COTTERELL, EDWARD, 5, West Halkin Street, S.W.
O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

ELECTED

1875 COUPLAND, SIDNEY, M.D. (C.), 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7.) *Trans.* 5, *C.S.* 2.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B. *Trans.* 2.

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 14.

O.M. CROFT, JOHN, 3, Mansfield Street, W. (C. 1870–2, V.P. 1882–4.) *Trans.* 12.

1890 CROWLE, THOMAS HENRY RICKARD, 3, Campden Hill Road, W.

1893 CURTIS, HENRY JONES, M.D., B.S., University College Hospital, W.C.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1891 DARDENNE, HENRI, M.B., 12, Torrington Square, W.C

1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.

1886 DAVIES, ARTHUR T., M.D., 23, Finsbury Square, E.C. *C.S.* 12.

1879 DAVIES-COLLEY, J. NEVILLE C., M.B., M.C., 36, Harley Street, W. (C. 1885–6.) *Trans.* 7, *C.S.* 1.

1879 Davy, HENRY, M.D., 29, Southernhay, Exeter.

1889 DEAN, HENRY PERCY, 84, Wimpole Street, W.

1879 Dennis, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.

1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6.) *Trans.* 5.

O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 9, Chesterfield Street, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1891 DICKINSON, WILLIAM LEE, M.D., 9, Chesterfield Street, W. *Trans.* 5.

1894 Dickson, THOMAS HUGH, M.B., B.C., 32, Belvedere Road, Norwood, S.E.

1871 DIVER, EBENEZER, M.D., Kenley, Caterham Valley, Surrey. (C. 1890–2.) *Trans.* 1.

O.M. DOWN, JOHN LANGDON H., M.D., 81, Harley Street, W. (C. 1870–2.) *Trans.* 1.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., 2, Manchester Square, W. (C. 1886–8.) *Trans.* 2.

O.M. DUCKWORTH, Sir DYCE, M.D., LL.D. (*President*), 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 18, *C.S.* 5.

O.M. DUFFIN, ALFRED B., M.D., 18, Devonshire Street, W. (C. 1872–4. V.P. 1891–3.) *Trans.* 6.

1884 DUKE, EDGAR, 30, Pevensey Road, St. Leonard's-on-Sea.

1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.

1889 DUNCAN, JOHN, M.D., St. Petersburg.

ELECTED

1889 DUNN, LOUIS ALBERT, M.S., 10, St. Thomas's Street, S.E. *Trans.* 1, *C.S.* 1.

1884 EDMUNDS, WALTER, M.C., 75, Lambeth Palace Road, S.E. *Trans.* 1.

1882 EMOND, EMILE, M.D., 113, Boulevard Beaumarchais, Paris.

O.M. ERICHSEN, JOHN E., LL.D., F.R.S., 6, Cavendish Place, W. (V.P. 1869–71.)

1868 EVANS, JULIAN, M.B., Marwood House, Honiton, Devon.

1893 EVANS, WILLMOTT H., 6, Gower Street, W.C.

1888 EVE, FREDERIC S., 125, Harley Street, Cavendish Square, W. *Trans.* 3, *C.S.* 4.

1877 EWART, WILLIAM C., M.D., 33, Curzon Street, W. (C. 1884–6.) *C.S.* 1.

1893 EZARD, EDWARD HENRY, M.D., B.S., 220, Lewisham High Road, S.E.

1868 Fairbank, FREDERICK ROYSTON, M.D., 16, Eversfield Place, St. Leonards-on-Sea. *Trans.* 2.

1889 FARDON, EDWARD ASHBY, Middlesex Hospital.

1885 FENN, EDWARD LIVEING, M.D., Grey Friars, Colchester.

1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.S.* 2.

1872 FENWICK, J. C. J., M.D., 25, North Road, Durham.

1893 FENWICK, WILLIAM SOLTAU, M.D., 10, Devonshire Street, W. *Trans.* 2.

1878 FIELD, GEORGE P., 34, Wimpole Street, W.

1876 FINLAY, DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 7.

1885 FITZ-PATRICK, THOMAS, M.D., 30, Sussex Gardens, Hyde Park, W.

1889 FLEMMING, PERCY, M.D., 93, Gower Street, W.C. *C.S.* 1.

1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W.

1878 FONMARTIN, HENRY DE, M.D., 26, Newberry Terrace, Lower Bullar Street, Nichol's Town, Southampton.

1889 FORBES, DANIEL MACKAY, Shoreditch Infirmary, 204, Hoxton Street, N.

1890 FORMAN, E. BAXTER, M.D., 11, Bramham Gardens, S. Kensington, S.W.

1890 FOSTER, MICHAEL G., M.B., M.A., Villa San Giovanni, Alassio, Italy.

1881 FOWLER, JAMES KINGSTON, M.D., 35, Clarges Street, W. (C. 1887–9.) *Trans.* 4, *C.S.* 5.

1886 FOX, R. HINGSTON, M.D., 23, Finsbury Square, E.C.

1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 6, *C.S.* 1.

1893 FOXWILL, ARTHUR, M.D., 7, Newhall Street, Birmingham.

1887 FREEMAN, HENRY WILLIAM, 24, Circus, Bath.

1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.

1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. *Trans.* 1.

1888 GAGE-BROWN, CHARLES HERBERT, M.D., 74, Cadogan Place, S.W.

1895 GALLOWAY, JAMES, M.D., 21, Queen Anne Street, W.

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, W. *C.S.* 1.

1879 GARSTANG, THOMAS WALTER HARROPP, The Heath, Knutsford, Cheshire.

ELECTED

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.

1893 GIBBS, CHARLES, Charing Cross Hospital.

1868 GLOVER, JAMES GRIN, M.D., 25, Highbury Place, N. (C. 1878–80, V.P. 1892–4.) *Trans.* 2.

1893 GLOVER, LEWIS G., M.B., B.C., Merton Lodge, West Hill, Highgate, N.

1882 GODDARD, EUGENE, M.D., 106, Highbury New Park, N.

1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895.) *Trans.* 12, *C.S.* 6.

1882 GOLDIE, ROBERT WILLIAM, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley.

1878 GOLDING-BIRD, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9.) *Trans.* 13, *C.S.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton. *Trans.* 3.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2.) *Trans.* 13, *C.S.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., 3, Grove Crescent, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, W.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895, S. 1892–4.) *Trans.* 11, *C.S.* 5.

1875 GOWERS, WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2.) *Trans.* 5.

1891 GRANT, J. DUNDAS, M.D., 8, Upper Wimpole Street, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.S.* 1.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 GRIFFITH, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1883 GROSS, CHARLES, M.D., M.S., 112, Westbourne Grove, W.

1895 GRUBE, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD, M.D., 15, Upper Berkeley Street, W.

1887 HABERSHON, SAMUEL HERBERT, M.D., 70, Brook Street, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–87.) *Trans.* 8, *C.S.* 4.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 Handford, HENRY, M.D., 14, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.S.* 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

ELECTED

O.M. HARLEY, JOHN, M.D., F.L.S., 9, Stratford Place, W. (C. 1875.) *Trans.* 1.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., The Infirmary, East Dulwich Grove, S.E. *Trans.* 1.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 HAWARD, J. WARRINGTON, 16, Savile Row, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 18.

1886 HAWKINS, FRANCIS HENRY, M.B., 26, Portland Place, Reading. *Trans.* 3, *C.S.* 3.

1889 HAWKINS, HERBERT PENNELL, M.B., B.S., 109, Harley Street, W. *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 162, Upper Parliament Street, Liverpool.

O.M. HEATH, CHRISTOPHER 36, Cavendish Square, W. (C. 1867–71, V.P. 1876–8, T. 1879–88, P. 1889–90.) *Trans.* 20.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1882 HERON, GEORGE ALLAN, M.D., 57, Harley Street, W.

1884 HERRINGHAM, WILMOT PARKER, M.D., 13, Upper Wimpole Street, W. (C. 1894–5.) *Trans.* 2, *C.S.* 2.

1888 Hetherington, GEORGE HAYNES, 10, Museum Street, Ipswich.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 Holman, CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–5.)

O.M. HOLMES, TIMOTHY, 6, Sussex Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) *Trans.* 16.

O.M. HOLTHOUSE, CARSTEN, Bath Terrace, Richmond. (C. 1870–2.) *Trans.* 8.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, W. (C. 1891–3.) *Trans.* 1.

1883 HOPKINS, JOHN, Central London Sick Asylum, Cleveland Street, W. *C.S.* 1.

1884 HORSLEY, VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.S.* 1.

1895 HOUGH, CHARLES HENRY, Fall Street, Derby.

1880 HOVELL, T. MARK, 105, Harley Street, W.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1876 HOWSE, HENRY GREENWAY, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2.) *Trans.* 3.

1894 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, 8, Queen Anne Street, W.

O.M. HUMPHRY, Sir GEORGE MURRAY, M.D., LL.D., F.R.S., Cambridge. (V.P. 1867–70.)

ELECTED

1892 HUNTER, WILLIAM, M.D., 54, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.S.* 1.

1879 INKSON, JAMES, M.D., Brigade Surgeon, Army.

1893 IONIDES, THEODORE HENRY, 30, Palmeira Square, West Brighton.

1883 JACKSON, GEORGE HENRY, "St. Levan's," Upperton, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2.

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., M.Ch., 66, Great Cumberland Place, W. (C. 1890–2.) *Trans.* 2, *C.S.* 1.

1888 JAMISON, ARTHUR, M.D., C.M., 18, Lowndes Street, S.W.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1875 JESSETT, FREDERICK BOWREMAN, 1, Buckingham Palace Mansions, S.W. *Trans.* 1.

1889 JOHNSON, RAYMOND, M.B., B.S., 20, Weymouth Street, W. *Trans.* 1.

1895 JOHNSON, Sir GEORGE, M.D., 11, Savile Row, W.

1893 JOHNSTON, G., M.B., 6, Manchester Square, W.

1878 JOHNSTON, WILLIAM, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, Harborne, Birmingham. *Trans.* 1.

1886 JULER, HENRY EDWARD, 23, Cavendish Square, W.

1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895.) *Trans.* 2.

O.M. KELLY, CHARLES, M.D., Worthing, Sussex.

1882 KESTEVEN, WILLIAM HENRY, Hillwood, Waverley Grove, Hendon, N.W. *Trans.* 2.

1883 KIDD, PERCY, M.D., 60, Brook Street, W. (C. 1891–2.) *Trans.* 4, *C.S.* 2.

1887 Knaggs, R. LAWFORD, B.C., Huddersfield. *Trans.* 1.

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead.

1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House, Swansea, S. Wales. *Trans.* 1.

1895 LANE, JAMES ERNEST, 45, Queen Anne Street, W.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 8, St. Thomas's Street, S.E. (C. 1893–5.) *Trans.* 23, *C.S.* 9.

O.M. LANGTON, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4.) *Trans.* 2.

1886 LANKESTER, HERBERT, M.D., Church Missionary House, Salisbury Square, E.C.

1885 LARDER, HERBERT, Whitechapel Infirmary, Baker's Row, E. *C.S.* 1.

1893 LAWSON, ARNOLD, 12, Harley Street, W.

O.M. LAWSON, GEORGE, 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1877 Lediard, HENRY AMBROSE, M.D., 41, Lowther Street, Carlisle. (C. 1889.) *Trans.* 5.

ELECTED

1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885.) *Trans.* 4.
1893 LENDON, EDWIN HARDING, M.B., 8, Norland Place, Holland Park, W.
1892 LEWIS, EDWARD JOHN, M.B., B.C., 87, Hamilton Terrace, N.W.
1895 LEWIS, ERNEST E., M.D., Springfield, Chelmsford.
1879 LICHTENBERG, GEORGE, M.D., 47, Finsbury Square, E.C.
1890 LITTLE, JOHN FLETCHER, M.B., 32, Harley Street, W. *C.S.* 2.
1868 LITTLE, LOUIS STROMEYER, China.
1891 LITTLEWOOD, HARRY, 40, Park Square, Leeds. *Trans.* 1.
1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, W.
1872 LIVEING, ROBERT, M.D., 11, Manchester Square, W. (C. 1883–4.) *Trans.* 2.
1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. *Trans.* 2.
1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 4, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.
1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.
1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5.) *Trans.* 9.
1894 LUFF, ARTHUR PEARSON, M.D., 31, Weymouth Street, W.
1879 LUNN, JOHN REUBEN, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1.) *Trans.* 7, *C.S.* 8.
1893 LYS, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.
1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.
1871 MAC CORMAC, Sir WILLIAM, 13, Harley Street, W. (C. 1877–9, V.P. 1888–9.) *Trans.* 5.
1891 MACDONALD, GRENVILLE, M.D., 85, Harley Street, W.
1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.
1882 MACKENZIE, FREDERIC MORELL, 29, Hans Place, S.W.
1891 MACKENZIE, HECTOR W. G., M.A., M.D., 59, Welbeck Street, W. *Trans.* 6.
1879 MACKENZIE, STEPHEN, M.D., 18, Cavendish Square, W. (C. 1884, 1888–90, S. 1885–7.) *Trans.* 9, *C.S.* 10.
1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath.
1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan Place, S.W. (C. 1889–91.) *Trans.* 8.
1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.
1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.
1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.
1881 MAKINS, GEORGE HENRY, (*Hon. Secretary*), 47, Charles Street, Berkeley Square, W. (C. 1892–4, S. 1895.) *Trans.* 2, *C.S.* 5.
1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. *Trans.* 1, *C.S.* 1.

ELECTED

1890 MANSON, PATRICK, M.D., C.M., 21, Queen Anne Street, W. (C. 1895.)
1888 Marriott. HYDE, M.B., Dial House, Stockport.
1868 MARSH, F. HOWARD, 30, Bruton Street, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8.) *Trans.* 12, *C.S.* 8.
1887 MARTIN, SIDNEY, M.D., B.S., 10, Mansfield Street, W.
1888 MASON, DAVID JAMES, M.D., C.M., Maidenhead.
1892 MASTERS, JOHN ALFRED, M.D., 57, Lexham Gardens, Kensington, W.
1884 MAUDSLEY, HENRY CARR, M.D., 11, Spring Street, Melbourne, Victoria,
1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.
1888 MAY, WILLIAM PAGE, M.D., B.Sc., 38, Weymouth Street, W.
1888 MENZIES, J. HERBERT, 47, Earl's Court Square, S.W.
1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Hospital for Sick Children, Moor Edge, Newcastle-on-Tyne.
1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.
1894 MICHELS, ERNST, 6, West Street, Finsbury Circus, E.C.
1873 MICKLE, WILLIAM JULIUS, M.D., Grove Hall Asylum, Bow, E.
1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.
1882 MONEY, ANGEL, M.D. (C. 1888–90.) *Trans.* 8.
1874 MORGAN, JOHN HAMMOND, 68, Grosvenor Street, W. (C. 1883–5.) *Trans.* 2, *C.S.* 6.
1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2.
1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6.) *Trans.* 9.
1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.S.* 1.
1885 MOTT, FREDERICK WALKER, M.D., 84, Wimpole Street, W. (C. 1895.) *Trans.* 1, *C.S.* 4.
1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 8, *C.S.* 2.
1875 MURPHY, SHIRLEY F., 22, Endsleigh Street, Tavistock Square, W.C. (C. 1888–90.) *C.S.* 1.
1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.
1893 MURRAY, GEORGE REDMAYNE, M.B., 2, Saville Place, Newcastle-on-Tyne.
1883 MURRAY, HUBERT MONTAGUE, M.D., 27, Savile Row, W. (C. 1893–5.) *Trans.* 1.
1894 MURRAY, JOHN, 133, Harley Street, W.
1868 MYERS, ARTHUR BOWEN RICHARDS, 43, Gloucester Street, Warwick Square, S.W. (C. 1877–9.) *Trans.* 1.
1872 MYRTLE, ANDREW S., M.D., 8, Park Parade, Harrogate. (C. 1892.)
1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.
1875 NETTLESHIP, EDWARD, 5, Wimpole Street, W. (C. 1881–2.) *Trans.* 2.
1889 Newman, D., M.D., 18, Woodside Place, Glasgow. *Trans.* 1.
O.M. NORTON, ARTHUR TREHERN, 101, Harley Street, W. (C. 1874–6.) *Trans.* 7.

ELECTED

O.M. NUNN, THOMAS WILLIAM, 8, Stratford Place, W. (C. 1873–4.) *Trans.* 8.

1880 O'CONNOR, BERNARD, M.D., 25, Hamilton Road, Ealing, W. *Trans.* 1.

O.M. OGLE, JOHN WILLIAM, M.D., 30, Cavendish Square, W. (C. 1867–8, V.P. 1884–5.) *Trans.* 6.

1868 Ogle, WILLIAM, M.D., 98, Friar Gate, Derby.

1883 OLIVER, GEORGE, M.D., West End Park, Harrogate. *Trans.* 1.

1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

1887 OPENSHAW, THOMAS HORROCKS, M.B., 16, Wimpole Street, W.

1868 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Germany. *Trans.* 1.

1877 ORD, WILLIAM MILLER, M.D. (*Treasurer*), 37, Upper Brook Street, W. (C. 1882–4, T. 1889–93.) *Trans.* 9, *C.S.* 1.

1890 ORD, W. WALLIS, M.B., B.Ch., 2, Queen Street, Mayfair, W. *Trans.* 1, *C.S.* 1.

1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W.

1884 ORMSBY, LAMBERT HEPENSTAL, M.D., 4, Merrion Square West, Dublin.

1883 ORTON, GEORGE HUNT, M.B., 1A, Campden Hill Road, Kensington, W.

1877 OWEN, ISAMBARD, M.D., 40, Curzon Street, W. (C. 1888–90.) *Trans.* 1.

1888 OXLEY, ALFRED RICE, M.D., Streatham Common.

1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4.) *Trans.* 6, *C.S.* 1.

1884 PAGET, STEPHEN, 57, Wimpole Street, W. *Trans.* 5, *C.S.* 2.

1873 PARKER, ROBERT WILLIAM, 13, Welbeck Street, W. (C. 1882–4, 1890–2, S. 1887–9, V.P. 1893–5.) *Trans.* 14, *C.S.* 7.

1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. *Trans.* 1, *C.S.* 1.

1890 PARKIN, ALFRED, M.S., 5, Albion Street, Hull. *Trans.* 1.

1894 PARKINSON, JOHN PORTER, M.D., 116, Broadhurst Gardens, N.W. *C.S.* 2.

1881 PASTEUR, WILLIAM, M.D., 4, Chandos Street, W. (C. 1891–3.) *Trans.* 2, *C.S.* 3.

1893 PATERSON, DONALD ROSE, M.D., C.M., 18, Windsor Place, Cardiff.

1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 PAUL, JOHN ERNEST, M.B., 43, Queensborough Terrace, W. *Trans.* 1.

1883 PAUL, JOHN LISTON, M.D., 43, Queensborough Terrace, W.

O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1886 PAYNE, JOSEPH FRANK, M.D., 78, Wimpole Street, W. *Trans.* 1.

1879 PEEL, ROBERT, 130, Collins Street East, Melbourne, Victoria.

1886 Penny, WILLIAM JOHN, Chandos Villa, Queen's Road, Clifton, Bristol.

1887 PENROSE, FRANCIS GEORGE, M.D., 4, Harley Street, W. *Trans.* 1, *C.S.* 1.

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1895 PHEAR, ARTHUR C., M.B., B.C., 123, Gower Street, W.C.

ELECTED

1874 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta Street, W.
1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.
1884 PHILLIPS, SIDNEY, M.D., 62, Upper Berkeley Street, W. (C. 1893–5.) *Trans.* 5, *C.S.* 1.
O.M. PICK, THOMAS PICKERING, 18, Portman Street, W. (S. 1874–7, C. 1878–80, V.P. 1885–6.) *Trans.* 4.
1885 PITT, GEORGE NEWTON, M.D., 24, St. Thomas's Street, S.E. (C. 1894–5.) *Trans.* 2, *C.S.* 4.
1883 PITTS, BERNARD, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 4.
1871 Playne, ALFRED, M.B., Maidenhead.
1884 POLAND, JOHN, 4, St. Thomas's Street, S.E.
1884 POLLARD, BILTON, 24, Harley Street, W. (C. 1895.) *Trans.* 2.
1868 POLLOCK, JAMES EDWARD, M.D., 52, Upper Brook Street, W. (C. 1878–80.)
1871 POORE, GEORGE VIVIAN, M.D., 30, Wimpole Street, W. (C. 1879–81.) *Trans.* 4.
1873 PORT, HEINRICH, M.D., 48, Finsbury Square, E.C.
1881 POWELL, H. A., M.A., 44, Sandgate Road, Folkestone.
O.M. POWELL, R. DOUGLAS, M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90.) *Trans.* 4.
1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.
1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. *Trans.* 1, *C.S.* 1.
1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.
O.M. QUAIN, Sir RICHARD, Bart., M.D., LL.D., F.R.S., 67, Harley Street, W. (C. 1867–9.)
1893 RAKE, ALFRED THEODORE, M,B., B.S., Guy's Hospital, S.E.
1895 RAMSAY, HERBERT MURRAY, Guards' Club, S.W.
O.M. RAMSKILL, J. SPENCE, M.D., 5, St. Helen's Place, E.C.
1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.
1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.
1891 REMFRY, LEONARD, M.D., 60, Great Cumberland Place, W. *Trans.* 1.
O.M. REYNOLDS, Sir JOHN RUSSELL, Bart., M.D., F.R.S., 38, Grosvenor Street, W. (C. 1867–8.)
1868 RICE, MICHAEL W., M.D. (C. 1876–8.)
O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.)
1877 RIVINGTON, WALTER, M.S., 95, Wimpole Street, W. (C. 1886–8.) *Trans.* 3.
1873 Roberts, DAVID LLOYD, M.D., 11, St. John Street, Manchester.
1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.
1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)
1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.

ELECTED

1885 ROBINSON, ARTHUR HENRY, M.D., Mile End Infirmary, Bancroft Road, N.E. *C.S.* 3.

1890 ROBINSON, GEORGE SOMERVILLE, Surgeon-Major, 13, Lupus Street, St. George's Square, S.W.

1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. *C.S.* 5.

1885 ROBSON, A. W. MAYO, 7, Park Square, Leeds. (C. 1893–5.) *Trans.* 12.

1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 13, Upper Wimpole Street, W. *Trans.* 1.

1888 ROPER, ARTHUR, Lewisham Hill, Blackheath. *Trans.* 1.

1889 ROSS, DANIEL MCCLURE, 76, Upper Berkeley Street, W.

1877 ROTH, BERNARD, 29, Queen Anne Street, W. *Trans.* 1, *C.S.* 4.

1890 ROUGHTON, EDMUND WILKINSON, 33, Westbourne Terrace, W.

O.M. ROUSE, JAMES, 2, Wilton Street, S.W. (C. 1875–7.) *Trans.* 2.

1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.

1887 RUTHERFOORD, H. T., M.B.

1885 RYLE, REGINALD JOHN, M.D., Green View, Hadley Green, Barnet.

1882 SAINSBURY, HARRINGTON, M.D., 63, Welbeck Street, W.

1893 SANSOM, ARTHUR ERNEST, M.D., 84, Harley Street, W. *Trans.* 1, *C.S.* 1.

1873 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta Street, W. (C. 1882–3.)

1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.S.* 2.

1886 SCOTT, ALFRED, 15, German Place, Brighton.

1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.

1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.S.* 1.

1869 SEDGWICK, LEONARD WILLIAM, M.D., 2, Gloucester Terrace, Hyde Park, W. (C. 1879–81.)

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.

1878 SERMON, FELIX, M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.S.* 3.

1884 SHARKEY, SEYMOUR, J., M.D., 2, Portland Place, W. (C. 1895.)

1889 SHAW, LAURISTON ELGIN, M.D., 10, St. Thomas's Street, S.E.

1876 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

1886 SILCOCK, ARTHUR QUARRY, M.D., M.S., 52, Harley Street, W. *Trans.* 4. *C.S.* 2.

1879 SKERRITT, EDWARD MARKHAM, M.D., Coburg Villa, Richmond Hill, Clifton, Bristol. (C. 1895.) *Trans.* 2.

1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.

1882 SMITH, E. NOBLE, 24, Queen Anne Street, W. *Trans.* 1.

1888 SMITH, FREDERICK J., M.B., 4, Christopher Street, Finsbury Square, E.C.

1884 SMITH, R. PERCY, M.D., Bethlem Royal Hospital, St. George's Road, S.E.

1893 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions, Baker Street, W.

Elected

O.M. Smith, Thomas, 5, Stratford Place, W. (C. 1869–71, V.P. 1880–2.) *Trans.* 14.

1875 Smith, T. Gilbart, M.A., M.D., 68, Harley Street, W. (C. 1883–5.)

1894 Smith Thomas Rudolph, M.B., B.C., 5, Stratford Place, W.

1872 Smith, William Wilberforce, M.D., 14, Stratford Place, W. *C.S.* 1.

1893 Snape, Ernest Alfred, 41, Welbeck Street, W.

1868 Snow, William V., M.D., Richmond Gardens, Bournemouth.

1890 Solly, Ernest, M.B., Strathlea, Harrogate, Yorks. *C.S.* 1.

O.M. Southey, Reginald, M.D., 32, Grosvenor Road, Pimlico, W. C 1867–70, 1876–8, S. 1873–5, V.P. 1883–4.) *Trans.* 16.

1888 Spencer, Walter George, M.S., M.B., 35, Brook Street, W. *Trans.* 2, *C.S.* 6.

1885 Spicer, Frederick, M.D., 282, Camden Road, N.W.

1888 Spicer, Robert Henry Soanes, M.D., 28, Welbeck Street, W. *C.S.* 1.

1882 Spooner, Frederick Henry, M.D., 4, Maitland Place, Lower Clapton, N.E.

1876 Squire, A. Balmanno, 24, Weymouth Street, W. *Trans.* 5, *C.S.* 4.

1892 Stabb, Ewen Carthew, St. Thomas's Hospital, S.E. *C.S.* 1.

1879 Staples, Francis Patrick, Brigade-Surgeon, Army.

1889 Stewart, Edward, M.D., Brook House, East Grinstead.

1874 Stirling, Edward C., M.D. [care of Messrs. Elder & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1888 Stoker, George, 14, Hertford Street, W. *C.S.* 1.

1881 Stokes, Henry Fraser, 2, Highbury Crescent, N.

1878 Stokes, Sir William, M.D., 5, Merrion Square North, Dublin. *Trans.* 2.

1884 Stonham, Charles, 4, Harley Street, W. *C.S.* 3.

1878 Strugnell, Frederick William, 45, Highgate Road, Highgate, N.W. *C.S.* 1.

1878 Sturge, William Allen, M.D., 29, Boulevard Dubouchage, Nice, France. *Trans.* 4.

1894 Sutherland, George A., M.D., 9, Old Cavendish Street, W.

1872 SUTHERLAND, Henry, M.D., 6, Richmond Terrace, Whitehall, S.W. *Trans.* 1.

1887 Sutton, John Bland, 48, Queen Anne Street, W. (C. 1894–5.) *Trans.* 10, *C.S.* 3.

1882 Symonds, Charters James, M.S. (C.), 26, Weymouth Street, W. (C. 1888–9.) *Trans.* 11, *C.S.* 4.

1876 Symonds, Horatio Percy, 35, Beaumont Street, Oxford.

1885 Tait, Edward Sabine, M.D., 48, Highbury Park, N.

1885 Tait, Henry Benwer, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 Tate, Walter William Hunt, 4, Queen Anne Street, W.

1886 Tay, Warren, 4, Finsbury Square, E.C.

1878 Taylor, Francis Thomas, M.B., 224, Lewisham High Road, S.E.

1875 Taylor, Frederick, M.D., 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–5.) *Trans.* 11, *C.S.* 3.

ELECTED

1889 Taylor, HENRY HERBERT, 10, Brunswick Place, Brighton.
1890 TAYLOR, JAMES, M.D., 34, Welbeck Street, W. *C.S.* 2.
1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.S.* 1.
1885 Taylor, W. C. EVERLEY, 34, Queen Street, Scarborough.
1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds.
1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.
1882 THIN, GEORGE, M.D., 22, Queen Anne Street, W. *Trans.* 1.
1886 THOMPSON, CHARLES HERBERT, M.D., Junior Constitutional, Piccadilly, W.
O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.
O.M. THOMPSON, Sir HENRY, 35, Wimpole Street, W. (C. 1867–8, V.P. 1886–7.) *Trans.* 3.
1894 THOMSON, ST. CLAIR, M.D., 28, Queen Anne Street, W.
1887 THORNTON, JOHN KNOWSLEY, M.B., C.M., 49, Montagu Square, W. (C. 1890–1.)
1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury. *Trans.* 5.
1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.
1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.
1892 TOOTH, HOWARD HENRY, M.D., 34, Harley Street, W.
1887 TOTSUKA, KANKAI.
1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.
1884 TREVES, FREDERICK, 6, Wimpole Street, W. *Trans.* 7. (C. 1893.)
1882 TURNER, FRANCIS CHARLEWOOD, M.D., 15, Finsbury Square, E.C. (C. 1887–9.) *Trans.* 1.
1882 TURNER, GEORGE ROBERTSON, 49, Green Street, W. *Trans.* 8.
1893 TURNEY, HORACE GEORGE, 28, Wimpole Street, W.
1888 TURNER, PHILIP DYMOCK, M.D., 95, Cromwell Road, S.W. *Trans.* 1.
1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8.) *Trans.* 6.
1881 UHTHOFF, JOHN CALDWELL, M.D., 46, Western Road, Hove, Brighton.
1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.
1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 31, Harley Street, W. *Trans.* 1.
1886 WADE, CHARLES H., Stanfield, Torquay.
1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)
1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., 67, Grosvenor Street, W. *Trans.* 2, *C.S.* 2.
1885 WAKLEY, THOMAS, jun., 5, Queen's Gate, W.
1885 WALKER, CHARLES ROTHERHAM, M.D., 7, Grove Road, Leytonstone, E.
1890 WALLIS, FREDERICK CHARLES, M.B., B.S., 26, Welbeck Street, W.
1875 WALSHAM, WILLIAM J., 77, Harley Street, W. (C. 1882–4.) *Trans.* 5, *C.S.* 1.

ELECTED

1888 WALTERS, FREDERICK RUFENACHT, M.D., 60, Welbeck Street, W. *C.S.* 2.
1888 Warner, PERCY, Woodford, Essex.
1894 WASHBOURN, JOHN WYCHENFORD, M.D., 15, Trinity Square, S.E. *Trans.* 2.
1891 WATERHOUSE, HERBERT FURNIVALL, M.D., 81, Wimpole Street, W.
1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., 30, Welbeck Street, W.
O.M. WEBER, HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.
1876 WEIR, ARCHIBALD, M.D., St. Mungho's, Great Malvern.
1868 WELLS, Sir THOMAS SPENCER, Bart., 3, Upper Grosvenor Street, W. (C. 1873.)
1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 12, *C.S.* 3.
1895 WETHERED, FRANK, J., M.D., 83, Harley Street, W.
1874 WHEELHOUSE, CLAUDIUS GALEN, Hilary Place, Leeds. *Trans.* 1.
1868 WHIPHAM, THOMAS TILLYER, M.D., 11, Grosvenor Street, W. (C. 1878–80, V.P. 1894–5.) *Trans.* 13.
1874 WHISTLER, W. MCNEILL, M.D., 17, Wimpole Street, W.
1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane Street, S.W.
1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.
1890 WHITE, GILBERT B. MOWER, M.B., B.S., 105, Gower Street, W.C.
1894 WHITE, JOSEPH, 6, Southwell Gardens, South Kensington, S.W.
1883 WHITE, WILLIAM HALE, M.D. (*Hon. Secretary*), 65, Harley Street, W. (C. 1887–9. S. June, 1893.) *Trans.* 10, *C.S.* 4.
1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *C.S.* 1.
1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.
1882 WHITTLE, EDWARD GEORGE, M.D., 65, Dyke Road, Brighton.
1871 WIGHT, GEORGE, M.B., C.M.; 428, Liverpool Road, N.
1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.
1894 WILKIN, GRIFFITH CHARLES, 39, Weymouth Street, W.
1884 WILLCOCKS, FREDERICK, M.D., 14, Mandeville Place, W. *C.S.* 1.
O.M. WILLETT, ALFRED, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.S.* 2.
1890 WILLETT, EDGAR, M.B., 25, Welbeck Street, W.
1888 WILLIAMS, CAMPBELL, 24, Welbeck Street, W.
O.M. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P, 1889–90.) *Trans.* 8.
1888 WILLIAMS, DAWSON, M.D., 25, Old Burlington Street, W. (C. 1893–5.)
1881 WILLIAMS, Sir JOHN, Bart., M.D., 63, Brook Street, W. (C. 1885–6.)
1890 WILLIAMS, W. ROGER, 28, Winckley Square, Preston.
1876 WILLIAMSON, JAMES MANN, M.D., Ventnor, Isle of Wight.
O.M. WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.

ELECTED

1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Bexhill, Hastings.
1889 WILLS, WILLIAM ALFRED, M.D., 23, Lower Seymour Street, W.
1886 WILSON, ALBERT, M.D., Leytonstone, Essex. *C.S.* 1.
1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. *Trans.* 2.
1891 WILSON, THEODORE STACEY, M.B., C.M., 65, Temple Row, Birmingham.
1890 WOOD, NEVILLE, 42, Elvaston Place, Queen's Gate, S.W.
1883 WOODCOCK, JOHN ROSTRON, Boston Spa, R.S.O. Yorkshire.
1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.
1894 WOOLLETT, CHARLES JEROME, 35, Telford Avenue, Streatham Hill, S.W.
1884 WORTS, EDWIN, 6, Trinity Street, Colchester.
1888 WYMAN, WILLIAM S., M.D., Red Brae, 18, Putney Hill, S.W.
1892 WYNTER, WALTER ESSEX, M.D., B.S., 30, Upper Berkeley Street, W.
1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the 1st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Sir Henry Acland, M.D., F.R.S.
James Andrew, M.D.
Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., M.D.
Bernard Edward Brodhurst.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
William Selby Church, M.D.
Edward Clapton, M.D.
John Couper.
John Croft.
William Howship Dickinson, M.D.
John Langdon Down, M.D.
Sir Dyce Duckworth, M.D.
Alfred B. Duffin, M.D.
John Eric Erichsen, F.R.S.
John Harley, M.D.
Christopher Heath.
Graily Hewitt, M.D.
Timothy Holmes.
Carsten Holthouse.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Sir William Jenner, Bart., M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Trehern Norton.
Thomas William Nunn.
John William Ogle, M.D.
Sir James Paget, Bart., F.R.S.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Richard Douglas Powell, M.D.
Sir Richard Quain, Bart., M.D., F.R.S.
J. Spence Ramskill, M.D.
Sir John Russell Reynolds, Bart., M.D., F.R.S.
Sydney Ringer, M.D., F.R.S.
James Rouse.
Thomas Smith.
Reginald Southey, M.D.
Edward Symes Thompson, M.D.
Sir Henry Thompson.
Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.B.
Christian G. H. Baümler, M.D.
John Cavafy, M.D.
James Grey Glover.
T. Henry Green, M.D.
1868 Howard Marsh.
Arthur Bowen Richards Myers.
Charles Prentis.
Edgcombe Venning.
Sir Thomas Spencer Wells, Bart.
John Ford Anderson, M.D.

1868 George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
William Ogle, M.D.
James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.
John Meaburn Bright, M.D.
Louis Stromeyer Little.

1869 Robert Brudenell Carter.
Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.

1871 Julius Althaus, M.D.
Robert M. Gover, M.B.
Sir William Mac Cormac.
Alfred Playne, M.B.
William F. Butt.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.

1872 Thomas Cooke.
J. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
Robert Liveing, M.D.
G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.
Henry Sutherland, M.D.
William Wilberforce Smith, M.D.

1873 William Julius Mickle, M.D.
Robert William Parker.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Heinrich Port, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.

1874 John Hammond Morgan.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Charles Douglas F. Phillips, M.D.

1874 W. M. Whistler, M.D.
Edward C. Stirling, M.D.
William Henry Bennett.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.

1875 Thomas Barlow, M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Frederick Bowreman Jessett.
Edward Liveing, M.D.
Edward Nettleship.
William J. Walsham.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
T. Gilbart Smith, M.D.
James Frederic Goodhart, M.D.
William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Shirley F. Murphy.
Herbert W. Page.
Frederick Taylor, M.D.

1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
Archibald Weir, M.D.
David White Finlay, M.D.
Henry Greenway Howse, M.S.
Furneaux Jordan.
R. Clement Lucas, B.S.
James Mann Williamson, M.D.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.

1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe Crocker, M.D.
David B. Lees, M.D.
Walter Hamilton Acland Jacobson, M.B., M.Ch.
Isambard Owen, M.D.
William Ewart, M.D.
Henry Morris, M.B.
William Miller Ord, M.D.
Walter Rivington, M.B.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton.
Malcolm Alex. Morris.

1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.

1878 Felix Semon, M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald Wm. Charles Hood, M.D.
Sir Joseph Lister, Bart., F.R.S.
Francis Thomas Tayler, M.B.
F. de Havilland Hall, M.D.
Storer Bennett.
Sir William Stokes, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
William Johnston, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.
Frederick William Strugnell.
1879 William Edward Burton.
James Magill, M.D.
Wm. John Vereker Bindon, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
James Inkson, M.D.
John Abercrombie, M.D.
F. G. Dawtrey Drewitt, M.D.
Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courteney Henderson, M.D.
Thomas John Maclagan, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.
George Lichtenberg, M.D.
Charles W. Mansell Moullin.
John Reuben Lunn.
Armand de Watteville, M.D.
George P. M. Woodward, M.D.
J. Neville Davies-Colley, C.M.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
John Williams, M.D.
Montagu Lubbock, M.D.
William Pasteur, M.D.
Henry Fraser Stokes.
John Caldwell Uhthoff, M.D.
1881 Henry Trentham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
William Henry Kesteven.
Frederic Morell Mackenzie.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Francis Charlewood Turner, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Eugene Goddard, M.D.
Charters James Symonds.
Angel Money, M.D.
C. F. Coxwell, M.B.
George Allan Heron, M.D.
Augustus Joseph Pepper, M.B.
Harrington Sainsbury, M.D.
George Thin, M.D.
Edwin Francis White.
1883 Charles Gross.
Anthony A. Bowlby.
Cecil Yates Biss, M.D.
Percy Kidd, M.D.
William Henry White, M.D.
George Oliver, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.B.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
William Hale White, M.D.
William Coode Adams, M.B.
William Anderson.
Robert Leamon Bowles, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
John Liston Paul, M.D.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.B.
John Hopkins.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.

1884 Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.B
Philip Henry Pye-Smith, M.D. F.R.S.
Charles Stonham.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.B.
Frederick Treves.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.D.
Stephen Paget.
Lambert Hepenstal Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard.
1885 Frederick Spicer, M.B.
Herbert Larder.
A. Hughes Bennett.
James Berry.
Frederick Walker Mott, M.D.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Philip Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Thomas Fitz-Patrick, M.D.
Henry Brewer Tait.
Charles Rotherham Walker, M.D.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.B.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne.
Edward Liveing Fenn, M.D.
1886 Thomas Dixon Savill, M.D.
John Cahill.
Charles Henry Wade.
Benjamin Wainewright.
1886 Waren Tay.
William John Penny.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.B.
R. Hingston Fox, M.D.
Henry Edward Juler.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Pridgin Teale.
H. H. Lankester.
Arthur T. Davies, M.B.
Charles Herbert Thompson, M.D.
Arthur Quarry Silcock.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.
1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton.
John Bland Sutton.
Oswald Auchinleck Browne, M.B.
Albert C. Butler-Smythe.
Joseph Arderne Ormerod, M.D.
C. J. Arkle, M.D.
J. H. E. Brock, M.B., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
Henry William Freeman.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S.
Thomas Horrocks Openshaw, M.B.
1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Arthur Roper.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.B.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
Arthur Jamison, M.D., C.M.
J. H. Menzies.

1888 Frank Ernest Roberts.
George Stoker.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. F. James, M.B.
Edwin A. Barton.
W. P. May, M.B.
Philip D. Turner, M.D.
William S. Wyman, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
George Haynes Hetherington.
David James Mason, M.D., C.M.
Walter G. Spencer, M.S., M.B.
1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. Davy Rolleston, M.B., B.S.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.D.
John E. Ranking, M.D.
William Alfred Wills, M.B.
Edward Ashby Fardon.
Wm. Alexander Carte, M.D., M.Ch.
Stanley Boyd, M.B.
George Ezra Halstead, M.D., B.S.
Edward Stewart, M.D.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.B., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.B.
Henry Percy Dean, M.B., B.S.
Louis Albert Dunn, M.S.
Percy Flemming, M.B.
Daniel Mackay Forbes.
H. Pennell Hawkins, M.B., B.S.
D. M. Ross.
Lauriston Elgin Shaw, M.D.
1890 John Rose Bradford, M.D.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
E. Baxter Forman, M.D.
1890 G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.
Thomas H. Rickard Crowle.
Robert A. Bindley.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.S.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Patrick Manson, M.D., C.M.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.B., M.A.
1891 Frederic François Burghard, M.D., M.S.
H. E. Leigh Cannev, M.B.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D.
Leonard Remfry, M.D.
Walter William Hunt Tate.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton
Henri Dardenne, M.B.
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.S., M.B.
Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.B., C.M.
1892 William Hunter, M.D.
Frank Thomas Paul.
Edward Cotterell.
Frank Richardson Blaxall, M.D.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.

1892 Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D., M.S.
Richard James Herbert Scott.
Howard Henry Tooth, M.D.

1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape.
Lewis G. Glover, M.B., B.C.
Solomon Charles Smith, M.D.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.B.
Donald Rose Paterson, M.D., C.M.
Edward Henry Ezard, M.D., B.Sc.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B, B.S.
Francis Charles Abbott, M.B., B.S.
James William Browne, M.B.
Edwin Harding Lendon, M.B.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber.
Theodore Henry Ionides.
Arthur Foxwell, M.D.
Horace George Turney.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.
G. Johnston, M.B.
Willmott H. Evans.
Charles Gibbs.

1893 Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.
Arnold Lawson.
Snape, Ernest Alfred.

1894 Herbert Morley Fletcher, M.D.
John Wychenford Washbourn, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels.
Charles Elliott Leopold Barton Hudson.
Charles Jerome Woollett.
St. Clair Thomson.
George A. Sunderland, M.D.
Joseph White.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.B., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B., B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.

1895 Herbert Murray Ramsay.
Arthur C. Phear, M.B., B.C.
Ernest C. Lewis, M.D.
Karle Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
Archie T. Collum, M.B.
Sir George Johnson, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1895.

THE COUNCIL is glad to be able to present a satisfactory report of the general prosperity of the Society.

The Members now number 546, of whom 25 have been elected during the Session. Fourteen Members have been lost during the year, four of whom—its President, Mr. Hulke; one of its Vice-Presidents, Sir George Buchanan; a late Vice-President, Mr. Arthur Durham, and Dr. Maunsell—have been lost by death. In reference to the death of its President, and at a Special Meeting of the Council summoned for the purpose, the following resolution was unanimously passed:—"The Council of the Clinical Society of London has learnt with deep regret of the death of the President of the Society, John Whitaker Hulke, F.R.S., one of its original Members, among its most earnest supporters, a highly-gifted and very learned surgeon, who has filled the office of President with his wonted punctuality, urbanity, and zeal. The Council begs to offer its heartfelt and respectful sympathy to Mrs. Hulke in her great sorrow." At the same meeting the Council appointed Mr. John Langton, the Senior Surgical Vice-President, Acting President for the remainder of the Session.

The new arrangements for the exhibition of clinical cases have worked well, and the three meetings devoted to this branch of the Society's work have been very largely attended and thoroughly successful.

A Committee of the Society has been appointed to investigate the clinical value of the Antitoxin of Diphtheria, and the late President nominated Dr. Church, Dr. Coupland,

Dr. Goodall, Dr. H. P. Hawkins, Dr. Sidney Martin, Dr. Stephen Mackenzie, Dr. Pasteur, Dr. Washbourne, and Dr. Hale White to serve on this Committee. The Committee is actively pursuing its investigations, but has not presented any Report to the Council.

The Council has nominated the following distinguished physicians and surgeons for election as Honorary Members of the Society:—Sir G. M. Humphry, M.D., F.R.C.S., LL.D., F.R.S.; J. Burdon Sanderson, M.D., F.R.C.P.Lond., D.C.L., LL.D., F.R.S.; Samuel Wilks, LL.D., M.D., F.R.C.P.Lond., F.R.S.; Dr. Carl Gerhardt; Dr. Theodor Kocher; Dr. Just Lucas-Championnière; Prof. H. Nothnagel; Prof. Rudolf Virchow, D.Sc.Cantab., D.C.L.Oxon.

The Treasurer's Statement of Account shows the finances of the Society to be in a very satisfactory condition.

THE CLINICAL SOCIETY OF LONDON.

Statement of Receipts and Payments for the Year ending 30th April, 1895.

WILLIAM M. ORD, Esq., M.D., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance at Bank, 1st May, 1894	357	10	2
223 Subscriptions at 21*s.*	234	3	0
170 „ received through Bank	178	10	0
23 Admission Fees at £2 2*s.*	48	6	0
1 „ Fee received through Bank	2	2	0
2 Composition Fees (Life)	31	10	0
1 „ Fee (Trans.)	5	5	0
Sale of *Transactions:*			
By the Publisher	39	17	11
Dividends on Consols* (£600)	7	19	6
	£905	3	7

PAYMENTS.	£	s.	d.
Cost of *Transactions*, Vol. XXVII:			
Paper, printing, binding, and delivery	187	0	3
Illustrations	99	13	9
Meetings:			
Expenses of rooms (Rent, &c.)	105	0	0
Refreshments	33	8	0
A. Tapson, for attendance	7	10	0
Treasurer and Hon. Secretaries:			
Secretarial assistance	26	5	0
Commission to Collector	15	15	0
Petty expenditure	6	0	9
Cheques	0	4	2
	480	16	11
Balance in hand	424	6	8
	£905	3	7

* *The sum shown here is only half of that actually accrued, but the remaining half lies at the Bank of England to the credit of the Society pending certification of death of the late Dr. Greenhow and of the appointment of his successor.*

JOHN LANGTON, *Acting President.*
WILLIAM M. ORD, *Treasurer.*

Examined with the vouchers and found correct.

SIDNEY PHILLIPS,
ANTHONY A. BOWLBY, } *Auditors.*

A. PEARCE GOULD, *Hon. Sec.*

May 9th, 1895.

BOSTON MEDICAL
DEC 6 1895
LIBRARY ASS'N.

COMMUNICATIONS.

I.—*A case of Aneurysm of the Basilar Artery.* By JOHN ROSE BRADFORD, M.D., F.R.S. *Read October* 12, 1894.

G. S., æt. 29, hospital porter, was admitted to University College Hospital at 8 P.M. on February 3, 1894.

On admission the patient was unconscious, but he had been seen on the preceding day by the Resident Medical Officer, and the following history obtained.

During the last fortnight he had complained of slight occipital headache and some stiffness of the muscles of the back of the neck. This he attributed to exposure to a draught. He was, however, sufficiently well to do his usual work as hospital porter until Friday, February 2. On Friday morning he ate for breakfast some bacon which he thought rancid, and subsequently he vomited and "felt out of sorts," but he did not at this time suffer from headache. He went to bed and felt better later in the day. Towards evening his wife said that he looked at her in a strange way, and "she thought he must have had a fit;" but on cross-examination no history of convulsions, change of colour, &c., could be obtained. He vomited three times in the course of the day. Pulse about 90, with occasional intermissions.

On February 3 patient was said to have had a fainting attack in the morning, and his wife stated that he stared in front of him, and did not recognise her. Vomiting had ceased, there was some occipital headache, bowels were constipated, pupils equal and of medium size, pulse still intermittent.

At 7 P.M. on February 3 patient was conscious and perfectly rational, the pupils were equal, but the headache was

rather worse. During the last twenty-four hours, however, retention of urine had set in, and patient was removed in an ambulance to hospital on account of this retention.

Subsequently it was ascertained that for six months previously patient had suffered from giddiness and noises in the head, and that seven years previously, whilst serving as a soldier in Africa, he had contracted syphilis, for which he had had no treatment.

Present state.—On admission at 8 P.M. patient was partially unconscious, but he could be roused sufficiently to open his eyes momentarily. The axillary temperature was 98° F. Pulse 70, irregular in force and intermitting about every fourth beat, incompressible. Veins not distended. Heart-sounds normal. Respiration natural and regular. Abdomen not retracted; no priapism. Urine drawn off, sp. gr. 1025, neutral, no albumen; reduces Fehling's solution, but no reaction was obtained with caustic potash. Patient is very restless, making constant attempts to get out of bed. There is probably some headache, as he occasionally puts his hand to his head. No tenderness of the scalp on percussion. The eyes deviate for the most part to the right, but occasionally they are directed straight forward; no movements of the eyes to the left were seen. Pupils medium in size, react to light, and dilate on stimulating the skin of the neck. No nystagmus was seen at any time. No paralysis or loss of sensation was observed in the face, but there was some retraction of the head. The tongue did not deviate as it lay in the floor of the mouth, but the patient could not be roused sufficiently to protrude it.

Although patient was constantly moving, the following position was the usual one. He lay on his left side, and there was some retraction of the head, but there was no spinal curvature. The thighs were flexed on the trunk, and the legs on the thighs, the feet forming a right angle with the legs. The arms were by the side of the trunk, and were slightly flexed; the hands were held prone, and the wrists were markedly flexed; the fingers were flexed at the metacarpo-phalangeal and extended at the interphalangeal joints. Abdomen was neither retracted nor distended. During the first hour after admission the patient was observed to roll over from left to right; this rolling movement was observed three times, and no such movements in the opposite direction were seen.

There were some rolling movements of the fingers and thumbs at the rate of five or six to the second. There was some

rigidity of the arms and legs, but the face was quite flaccid. No fibrillary twitchings were seen.

The legs were occasionally fully extended. Sensation to pain seemed present everywhere.

Reflexes.—(1) Superficial: the epigastric and abdominal were present, the plantar were notably increased.

(2) Deep: jaw, wrist, elbow, and knee-jerks were all greatly increased, and slight ankle-clonus was present, but no other clonus was elicited.

No involuntary micturition or defæcation occurred, and no vomiting.

Respiration was natural, and the chest and abdominal movements were synchronous. No attempt at articulation was made.

At 9 P.M. patient became quieter and the pulse increased to 100, and he lay quietly on his back. The eyes were directed straight forward, and the limbs became lax. The respirations became infrequent and sighing, and at about 10 P.M. it became necessary to perform artificial respiration. Priapism was now well marked, knee-jerks absent, and the pupils did not react to light. Pulse 110. Rectal temperature 99·2°. Artificial respiration was kept up for four hours, the pulse increased to 150 and became weaker, and as no attempt at spontaneous respiration occurred, the artificial respiration was stopped at 2.10 A.M. The pulse then became less and less frequent, and ceased two minutes later. No convulsions occurred. Temperature at midnight was 97° F. in the rectum.

Post-mortem examination thirty-two hours after death. Body well nourished. Scar on prepuce.

Heart and lungs normal. Aorta showed slight atheroma.

Liver: capsule much thickened in places, and there were numerous depressed cicatrices over the whole of the surface, especially of the left lobe. On section there was increase of fibrous tissue, and several gummata were found scattered through the organ.

Kidneys, intestines, and spleen normal.

Brain: the convolutions were flattened. There was no thickening of the meninges. On the under surface of the pons there was a mass of clot surrounding the basilar artery, and extending upwards on the sides of the medulla and pons, and also to a slighter extent on the under and upper surfaces of the cerebellum. The clot on the under surface of the pons was about a quarter of an inch in thickness; on the upper

surface of the cerebellum it was very thin. On section a small amount of clot was found in the third ventricle and still less in the lateral ventricles; the *iter*, however, and the fourth ventricle were distended with clot. On dissection a fusiform aneurysm of the basilar artery was found in the midst of the mass of clot, about three eighths of an inch long and a quarter of an inch in diameter.

No other vessel was found aneurysmal, and there was no blood in the subdural space.

I have brought this case before the Society owing to the long survival, *i. e.* about forty hours from the onset of symptoms, and also as illustrating the apparently trivial character of the initial symptoms, *i. e.* slight sickness and headache with slight irregularity of the pulse followed later by retention of urine. The case also shows the occurrence of blood-clot in the ventricles from extension from a hæmorrhage at the base of the brain. The blood extravasated from the ruptured aneurysm reached the ventricles by extending up the sides of the medulla and so into the fourth ventricle, and subsequently reaching the others through the iter.

On reviewing the symptoms it is probable that the giddiness and noises in the head during the last six months of life were caused by the aneurysm. The rupture of the aneurysm caused the initial symptoms of headache, vomiting, and irregularity of the pulse, but the extravasation of blood being small, life was not at once destroyed. Later, the gradually increasing extravasation (or perhaps a further rupture dependent on moving the patient) caused the cessation of respiration from extension of the clot to the fourth ventricle.

II.—*Two Cases of Intra-cranial Aneurysm in Young Adults, unaffected with malignant endocarditis or syphilis.* By W. Hale White, M.D. *Read October* 12, 1894.

MARY C., æt. 34, admitted into Guy's Hospital December 29, 1893. *Previous history.*—Has always been subject to severe headaches. Three months ago she had an abscess in her right ear; this burst. She was advised by Mr. Purves not to go far from medical advice.

Present illness.—On December 25, while at dinner, she suddenly fell off her chair, was picked up unconscious, and remained so eight minutes, the conjunctivæ being insensitive, the pupils normal, the body and limbs rigid. On coming round she had intense headache, and was violently sick, five times in half an hour, and occasionally afterwards. The headache, which was very severe, as though the head were being split open, prevented sleep, and has continued since the fit. On December 27 first complained of aching in the left ear.

On admission.—Intense headache, increased by light, and therefore she lies with her eyes shut. She cannot bear the weight of an ice-bag on her head; tongue furred, bowels constipated, no discharge from the ears. Pulse 60, resp. 20, temp. 100·4°; left optic disc hyperæmic and a little indistinct.

December 30.—Head retracted, great pain in occipital region, running down the spine to the sacrum; complains of pain about the left ear; much nausea, vomited once.

December 31.—Pain as yesterday, the membrane of the left ear looks healthy. Left optic disc as yesterday. No tenderness in mastoid region on either side; sick once.

January 1.—Pulse 60, temp. 100·4°, resp. 20.

January 2.—The pain down the back is very severe. The retraction of the head is less, there is slight right facial paralysis. This is said to have existed before the illness began. There is slight diplopia. The headache is most intense in the left temporal region. Pulse 70, resp. 22, temp. 100·4°.

January 3.—Is better in all respects, but left ptosis is very marked. Pulse 74, resp. 20, temp. 99·6°.

January 5.—Still improving. Hyperæmia and mild blurring of left disc less, ptosis less, but there is slight

left external strabismus. Pulse, temperature, and respiration normal.

January 6.—Not so well. More pain and throbbing, especially in the left temporal region.

January 7.—Headache very severe, especially over left temple; complete paralysis of left third nerve. Pulse 60, resp. 20, temp. 98·4°.

January 8.—Headache, especially left side, very intense. Left pupil does not react to light or accommodation. Had a slight fit, in which the face flushed, the right eye turned up, and the teeth were tightly fixed. Much hyperæsthesia down the spine. In the evening the right mastoid antrum was explored, but found to be healthy. Pulse 60, resp. 16, temp. 97·8°.

January 11.—Has been much the same. Headache and hyperæsthesia down the spine still very marked. Pulse 64, resp. 16, temp. 100°.

January 14.—The headache and pain down the spine are worse. Pulse and respiration irregular, temp. 100·6°.

January 15.—The head is retracted again. Had a fit like the last. Pulse 100, resp. 14, temp. 101·4°.

January 16.—Is delirious and semi-conscious. The arms and face twitch a good deal. Pulse 108, resp. 24, temp. 101°.

January 17.—Still unconscious. Much twitching of face and arms. Pulse 148, resp. 32, temp. 104·6°.

January 18.—Quite unconscious. At 1 A.M. suddenly became worse. Respiration slowing down to 4 to the minute, pulse became running, and she died in ten minutes. The pulse continued after the respiration had stopped.

During her illness she was given $\frac{1}{8}$ gr. of perchloride of mercury subcutaneously every night, morphia frequently for the headache. Local applications, such as leeches, were used, but they did not relieve the pain.

Post-mortem.—The dura mater and sinuses were healthy. Upon removing the brain there was a large amount of recent clot on the under surface in the subdural space, and a less amount of clot in the subarachnoid space. The clot had extended into the fourth ventricle, where it formed a cast of the cavity. The fluid in the ventricles was blood-stained. There was a layer of clot quite an eighth of an inch thick all down the spinal cord in the subarachnoid space. All the arteries and cerebral tissues were normal, except that there was an aneurysm, $\frac{7}{8}$ inch long and $\frac{1}{2}$ inch in diameter, which sprang from the left internal carotid just before its bifurca-

tion. This pressed on the left third nerve, and lay alongside the posterior communicating artery without implicating it. There was a large aperture in the aneurysm, its wall was thick, and had two patches of atheroma in it. The heart was quite healthy, and there was no evidence of syphilis.

John W., æt. 21, labourer, was admitted into Guy's Hospital January 23, 1894.

For the last few weeks he had been complaining of pain of a neuralgic nature in the right frontal region.

Present illness.—That morning, January 23, at 1 P.M., while carrying a sack of coals, he was observed to suddenly fall down. This was not because he stumbled, nor because the sack caused his fall; he was not observed to twitch the face or limbs. He soon came round, when he was slightly sick. He was then put into a cart to be taken home. Being there seized with another attack, in which the limbs were rigid, he was brought to the hospital.

On admission.—When brought into the surgery he was unconscious, the pupils were fixed and dilated, the eyeballs were prominent. There was some right external strabismus. The house physician thought him to be suffering from post-epileptic coma, but as he did not recover he was taken into the ward at 6 P.M. He was then noticed to be well nourished. He lay on his back without moving, breathing with stertor. Limbs were quite rigid, extended, and slightly adducted. From time to time there were slight spasms in which the adduction was more marked, and the back was slightly arched, and the jaw became rigid. The eyes were in the same condition as above described. There was no optic neuritis nor atrophy, but on the outer side of the right disc was a large prominent, dark brick-red, subretinal swelling. This encroached a little on the disc, and was four times the size of it. It was thought to be subretinal hæmorrhage. A similar swelling was seen in the left eye. *Reflexes.*—On admission both plantar and both knee-jerks were present, left knee-jerk was increased; left ankle-clonus, right was absent. Elbow- and wrist-jerks were present on both sides. 10 P.M., both knee and plantar greatly increased, ankle-clonus obtainable both sides. Elbow- and wrist-jerks violent. Throughout no conjunctival, cremasteric, abdominal, nor epigastric reflexes could be obtained. *The urine.*—On admission at 6 P.M. the bladder was greatly distended, two pints of urine were drawn off; sp. gr. 1005; copper test gave 2·5 per cent.

sugar; this was confirmed by yeast and phenyl-hydrazin tests. Shortly after this was drawn off the urine began to flow away; that collected at 8.30 P.M. contained only 1 per cent. of sugar. Some obtained post mortem contained no sugar. Between admission and death more than six pints were passed and drawn off. There was no albumen, and it was acid. *Pulse* slow, 52, at 6 P.M. Its frequency gradually increased; at 1 A.M. it was 132. It went on beating about half an hour after the final stoppage of breathing at 1.30. *Respiration*, 24 on admission, remained regular till 1 A.M., when it became irregular and rapid (44). It stopped at 1.30, and he became cyanosed. Artificial respiration was then tried for half an hour without effect. The diaphragm was never paralysed. *Temperature* equal on the two sides, 96°, rising to 97° at 8.30. At 8.30 the rigidity of the limbs passed away, the spasms ceased, nor could they be induced reflexly by stimuli, except in the left arm. The face was flushed, the feet were very cold.

Post-mortem.—On removing the skull blood-clot was found in both the subdural and subarachnoid cavities. It was all about the base of the brain, and there was a good deal on the upper surface. The hæmorrhage had passed forwards in the sheath of the optic nerves, which were much distended with blood-clot, and ultimately tore its way forwards under the retina. There was no blood-clot in the vertebral canal. The source of the hæmorrhage was an aneurysm, the size of a small marble, which sprang from the right internal carotid artery just before its termination. The aneurysm contained some old blood-clot, and its walls were atheromatous. All the other cerebral vessels were quite healthy, so were all the vessels in the rest of the body, every organ of which was perfectly healthy.

I thought these two cases worthy of the notice of the Society because of the extreme rarity of aneurysms in the first half of life apart from malignant endocarditis or syphilis.

In the first case the fact that the patient had had an abscess in the right ear, and complained of intense pain about the left ear, made us strongly suspect meningitis associated with ear disease. During life the occipital headache and intense pain and tenderness down the spine, together with well-marked retraction of the head, were also very suggestive of meningitis. Probably judged by the result of the post-mortem we ought to read the case thus. That the woman

had an aneurysm which up to December 25 gave rise to no symptoms; then it suddenly leaked, causing the sudden onset of a fit, followed by loss of consciousness and vomiting. Somehow this leaking ceased, but the effused blood gave rise to the headache, the rise of temperature, and the slow pulse, and passing backwards and down the spine caused the head to be retracted, and the pain and tenderness down the spine. As the clot became absorbed the patient improved, but meanwhile the aneurysm was growing, and to this was due the paralysis of the left third nerve and the pain on the left side of the head.

On January 15 a second rupture took place, and the effused blood caused the fatal coma and the rise of temperature to 104·6°. It is interesting to observe that this also was a slow leaking, for the patient did not die till January 18.

Some parts of the clot at the post-mortem examination certainly appeared to be of an earlier date than the greater quantity, but it was impossible to be positive on this point.

In the second case I would particularly direct attention to the hæmorrhage which could be seen during life under the retina in both eyes, and to the excessive secretion of urine. Glycosuria in meningeal hæmorrhage is not very surprising, but the fact that the specific gravity of the urine was only 1005 in spite of containing sugar shows how excessive must have been the excretion of water from the kidneys.

III.—*A case of Acute Pancreatitis.* By J. E. Paul, M.D. *Read October* 12, 1894.

C. B., male, æt. 43, walked into the Out-patient Department of University College Hospital at 12 midday on August 30, 1893, and complained that he was suffering from constipation.

The history he gave was the following. His bowels were opened on the 26th of August, but not being opened on the 27th he took a pill that evening to procure an evacuation. The pill failed to act; he went to his bed feeling quite well, but was wakened in the early morning with a sharp pain in the abdomen; the pain was not so severe as to make him cry out or roll about. The pain passed off in the course of a few minutes, and he went to sleep again.

On Tuesday, August 29, he had a return of the pain in the abdomen, but "nothing to speak of," as he said. He vomited twice on the 29th. He continued at his work until August 29.

On the night of August 29 he again took a purgative pill, but obtained no relief.

August 30.—Vomited once in the morning; had very slight pain in the abdomen. The constipation still persisted.

He came to the hospital on August 30.

Previous history.—Has had good health all his life; has had no serious illnesses and no previous attacks like the present. Says he is a moderate drinker, taking two pints of beer a day. No history of syphilis.

On examination.—Patient is a healthy-looking man, tending to be rather obese; answers all questions rationally; does not appear to be in pain; has a few dilated venules on the face. Conjunctivæ dull, slightly yellowish. Tongue slightly furred. Pulse 82, fairly full, slightly high tension, regular; temp. 100·6°.

Abdomen.—Abdominal walls normal. Fair amount of fat. Umbilicus depressed; no dilated veins to be seen; the abdomen is full and slightly distended.

Movement.—No diminution of movement, regular and equal on the two sides. Abdomen can be readily manipulated without causing the patient any pain. There was some resistance in the left flank over the region of the descending

colon; over this same area the percussion note was impaired. This was not altered by rolling patient over on to the opposite side. Percussion note over rest of the abdomen normal. The resistance and alteration of percussion note over the area of descending colon was thought to be due to fæces. No signs of fluid in abdomen.

Rectal examination.—Rectum loaded with fæces, otherwise normal.

Patient was advised to come into the hospital, at which he expressed great surprise, as he felt perfectly well except for the constipation.

On admission he was put to bed, and was given an enema of castor oil ℥j, and soap and water Oj, at 3 P.M. This brought away a very large constipated stool; no blood or slime in the stool. About 4.30 P.M. he was noticed to be collapsed, his extremities cold and clammy. Radial pulse only just perceptible; temperature 102·6°. On being asked if he had any pain, he said he had none, and said he felt all right. Manipulation of abdomen caused no pain; physical signs in abdomen as before; the dulness in left flank less marked.

The collapse gradually increased, and patient died at 10.30 P.M., eight hours after admission. Temperature just before death 104° F. There was no vomiting while patient was in the hospital.

Shortly before death patient passed about two drachms of urine. This was somewhat opaque, acid in reaction, and contained about a quarter albumen. No sugar. Under the microscope a few red corpuscles and hyaline and granular casts were seen.

Autopsy twelve hours after death. Post-mortem staining well marked. On opening the abdomen nothing abnormal was to be seen. On raising the intestines about ℥ij of an opaque fluid were seen in the pelvis. Intestines looked healthy, no injection, no lymph; they were not distended. On lifting up the stomach, the posterior surface was seen to be glued to the peritoneum covering the pancreas by thin recent lymph. The peritoneum covering the pancreas was thickened and œdematous.

The stomach on being opened was found to be healthy, no signs of irritation. No perforation, nor was anything abnormal detected in duodenum or intestines.

Pancreas: on the peritoneal surface was a small quantity of recent lymph. The head of the pancreas was normal, but

the rest of the organ was considerably swollen. On making a section through the pancreas, extending from the middle to the tail of the pancreas were seen necrotic patches with small hæmorrhages, the hæmorrhages varying in size from a pin's head to a split pea. These hæmorrhages were most marked in the tail of the pancreas. No abscess was found in the pancreas, no blood in the peritoneum or subperitoneal tissues. Kidneys normal except that the tubules seemed to be unduly injected. Mesentery normal, no fat necrosis. Other organs healthy.

Microscopical examination.—Pancreas: on examination of the section the glandular structure of the organ was only made out with some difficulty, the acini showed a considerable amount of disintegration, and in parts they were completely destroyed. Small hæmorrhages were seen dotted through the section, also a considerable amount of blood-pigment. No micro-organisms could be detected.

Kidneys showed well-marked cloudy swelling, otherwise normal. No micro-organisms detected. Other organs healthy.

IV.—*A Fatal Form of Tetany associated with chronic dilatation of the stomach.* By W. Soltau Fenwick, M.D. *Read October* 12, 1894.

Case 1.—A man æt. 34 years was admitted into the London Hospital on February 26, 1893, under the care of Dr. Smith, suffering from severe vomiting, with cramps in the arms and legs.

The patient was a carpenter by trade, and had always enjoyed good health until six months previously, when he began to experience pain in the stomach after his meals, accompanied by nausea and often by vomiting. There was no family history of any importance. He had never been a heavy drinker, and had never suffered from syphilis. He stated that two days before admission (February 24) he was suddenly attacked with violent vomiting, and a few hours afterwards with severe cramps in the hands and legs, which prevented him from walking.

On admission the patient was found to be a well-built and well-nourished man. The lips and cheeks were slightly congested, the temp. 97° F., the pulse weak but regular, 80 per minute, and the respirations 24 per minute. The arms were slightly abducted, the elbows flexed, and the fingers bent at the metacarpal and phalangeal joints. The thumbs were also flexed and adducted toward the median line of the palm. The legs were extended upon the thighs, and the soles of the feet somewhat inverted, with extension of the ankle-joints. Considerable pain was experienced when attempts were made to alter the position of the affected limbs. Both the plantar reflexes and knee-jerks were exaggerated. The bowels had not been opened for two days, nor had any urine been voided for the same period of time. On passing a catheter seven and a half ounces of dark-coloured urine, containing a trace of albumen, was drawn off. No abnormal physical signs could be detected in the chest, and the heart-sounds were clear. The pupils were contracted, but reacted slightly both to light and accommodation. The fundi were normal. The abdomen appeared to be generally distended, and was painful on pressure. The lower border of the stomach reached to the umbilicus, and a distinct splash was audible on manipulation. The patient vomited several times

within the first few hours of his residence in the hospital, the ejecta being bile-stained, exceedingly sour-smelling, and of considerable quantity.

The following day the pulse was of fair volume and regular, 76 per minute. There were no signs of dyspnœa, but slight cyanosis was noticeable. The bowels had reacted to a dose of elaterium, but the patient still suffered from retention of urine. When the bladder had been emptied by means of a catheter the fluid was found to contain a trace of albumen. The tonic spasm of the muscles of the arms and legs still continued, and frontal headache was complained of. There was no alteration in the sensibility of the skin of the affected parts, and the intellect was quite clear. The vomiting was still troublesome.

The next day (March 1) the cramps had disappeared, and the patient declared himself much better. In the evening, however, he was again attacked by severe spasm in the muscles of the extremities, preceded as on the former occasion by profuse vomiting. He also complained of occasional twitchings in the muscles of the face and mouth. The pupils were contracted, but reacted both to light and to accommodation. The knee-jerks and superficial reflexes were exaggerated. The skin over the outer side of the left thigh and leg was slightly hyperæsthetic. This attack of spasm lasted several hours, and finally disappeared as suddenly as it had shown itself.

On March 4 the spasms recurred while the patient was under observation. The fingers of both hands were firmly flexed, and pressed over the thumbs toward the palm of the hand. The legs became rigidly extended and adducted, while the feet assumed the position of equino-varus. The patient complained of great pain in the affected muscles, but there was no elevation of the temperature nor any modification of cutaneous sensation to be observed. The attack subsided in about fourteen hours, leaving the patient in a weak and very drowsy condition. The urine was retained during the continuance of the spasm, and when finally voided was found to contain a trace of sugar but no albumen.

During the next ten days the patient enjoyed a respite from his disease. He could move his legs easily, and walk with comfort. The urine was passed in a voluntary manner, and appeared to be perfectly normal. The reflexes, both superficial and deep, were less exaggerated than previously.

On March 14 the patient was seized with severe vomiting,

and a few hours later the tonic spasms again made their appearance, and lasted for an hour and a half.

Three days later the tetany exhibited itself for the fifth time, preceded as usual by severe vomiting. This attack was accompanied by intense vertical headache, but only lasted for a short time.

From the 17th to the 31st of March the patient remained free from nervous symptoms. He complained, however, of pain in the epigastrium after meals, and on one or two occasions he vomited his food. The limbs were quite free from rigidity, and he could walk about the ward.

On April 4 vomiting recommenced, and several pints of bilious fluid were ejected during the course of a few hours. The tonic spasm of the extremities returned quite suddenly, and at the same time the patient complained of stiffness of the face and neck. The urine was found to contain a trace of albumen, when withdrawn by a catheter, and the pupils were contracted to the size of pin-holes. During the course of the next few hours the pain and stiffness in the muscles of the face increased, and the jaw became affected in a similar manner, so that great difficulty was experienced in swallowing. The respiration became jerky in type, the temperature rose to 102° F., and the patient succumbed to respiratory failure about twelve hours after the onset of his seventh attack of tetany.

At the necropsy the stomach was found to be considerably dilated, and in close proximity to the pylorus there was a chronic ulcer, which by its contraction had given rise to partial stenosis of the orifice. The brain and kidneys were apparently healthy, and no other visceral disease could be discovered.

The case which has just been narrated was originally admitted into the hospital as an instance of kidney disease with uræmic convulsions; but the peculiar character of the spasms, combined with the absence of cerebral symptoms and any direct evidence of Bright's disease, were considered sufficient to negative the provisional diagnosis, and the case was subsequently regarded as an example of tetany. But, unlike ordinary tetany, the spasmodic attacks were invariably ushered in by severe vomiting, during which several pints of fluid were often ejected, while in the intervals of the disease the patient suffered much from epigastric pain, and presented the signs of a dilated stomach. It will also be remembered that his previous history indicated the probability of gastric

ulceration being present. This curious association of tonic spasm of the muscles of the extremities with severe gastric symptoms would in all probability have escaped notice, had it not been for the fact that Kussmaul, in the year 1869, had published three cases of a similar nature, two of which proved fatal. Since that time the so-called "tetany of gastric origin" has attracted considerable attention on the Continent, and in consideration of the great practical interest which is attached to this unusual complaint I would venture to offer a few remarks concerning its clinical symptoms and treatment.

In addition to Kussmaul's original cases I have been able to collect the records of twenty-three instances of the disease, making in all a total of twenty-six. Of this number twenty were males and only six females. The average age at which the disease appeared was forty-eight, the youngest patient being twenty-six and the eldest sixty-six. In the great majority of the cases the symptoms of gastric disease had existed for many years prior to the onset of the nervous affection, and in only two instances out of the entire number for a period of less than twelve months. Out of fourteen cases where an accurate account of the post-mortem examination is recorded, no fewer than ten exhibited a dilated stomach due to the contraction of a chronic ulcer in the pyloric region. In two cases the gastric dilatation was dependent upon the cicatrisation of a simple ulcer in the first portion of the duodenum; in one instance cancer of the pylorus co-existed with chronic ulcer, and in only one case was the increase in the size of the organ found to depend upon simple atony of its walls. In addition to these facts, three cases which ended in recovery suffered from repeated attacks of hæmatemesis, so that it is obvious that in at least 61 per cent. of the cases the dilatation of the stomach was a long-standing affection, and owed its origin to the presence of a chronic ulcer in the immediate vicinity of the pyloric orifice.

With only two exceptions a severe attack of vomiting invariably preceded the onset of the nervous symptoms, and in most instances proved so marked a feature in the case that Kussmaul was induced to regard it as standing in causal relationship to the convulsive seizures.

Although most of the writers on the subject have referred to the disease as a variety of tetany, a careful consideration of the clinical features presented by the various cases is sufficient to demonstrate the fact that a tonic contraction of the mus-

cles of the extremities is not the only symptom of the disease, but that general convulsions of a tetanic or epileptiform nature are extremely apt to supervene.

The nervous symptoms should therefore be divided into three classes:

1. A form of tonic contraction of the muscles of the hands, forearms, feet and legs, closely allied in its general features to true tetany.

2. An intermittent form of spasm attacking the muscles of the trunk, especially those of the jaw, neck, back, and respiratory system.

3. General convulsions of short duration, closely resembling ordinary epileptic fits.

These last two varieties never occur alone, but always in association with the first mentioned.

Attacks of tetany similar to those which occurred in the case I have cited at length were noted in every one of the twenty-six instances of the disease, and it must therefore be allowed that this variety constitutes the fundamental type of the disorder. It usually exhibits itself shortly after an attack of vomiting, and is solely confined to the muscles of the extremities. In the most typical instances the elbows are slightly flexed, the forearms pronated, and the arms adducted to the side. The fingers of the hands are firmly bent over the thumb, which is adducted toward the middle line of the palm. Considerable pain is often experienced during the continuance of the spasm, and in many instances the affected limbs appear blue, and are perceptibly cold to the touch. The condition of the superficial reflexes is variable, but the deep reflexes are usually much exaggerated, and in many cases the muscles react more readily than normal to the interrupted current. In some cases it was noticed that a spasm could be induced either by percussing the skin over the epigastrium, by compressing the main nerve or artery of the limb, or by the administration of an enema. In not a few instances the first attack appeared to follow an attempt to wash out the dilated stomach.

The other phenomena associated with this condition are neither uniform nor of great importance. The pupils are often contracted, but this is by no means invariable. Severe headache is a frequent symptom, and not infrequently profuse perspirations are a noticeable feature of the case. Retention of urine is often observed as an accompaniment of the attack, and when the urine is drawn off it is sometimes found to

contain either albumen or sugar. Cutaneous sensibility rarely undergoes any decided alteration, but in two of the recorded cases transient hyperæsthesia was observed. The pulse is full and regular, the breathing quick and generally shallow, and the face and extremities occasionally show signs of cyanosis. The temperature of the body remains at the normal point during an attack, and the intellect is unaffected.

In the majority of the cases the first attack is followed within a short period of time by several others, but occasionally the initial seizure is separated from the second by an interval of several days, or even months. The actual duration of the spasm is also liable to considerable variation, lasting in some cases from five minutes to three or four hours; while in others, again, it does not subside for several days.

Of the sixteen cases of simple tetany, eight (or 50 per cent.) ended fatally, death being ushered in by delirium, a quick pulse, a rapid rise of temperature, and finally by coma with dilated pupils.

Such is a brief outline of the various symptoms which usually present themselves during the course of the disease; and it will be noticed that one of the great distinctions between this form of tonic spasm and the disorder known as tetany lies in the extremely fatal nature of the former complaint. But I would here venture to suggest that slight instances of tetany associated with chronic dilatation of the stomach are really much more common than is at present believed, and in support of this statement would refer to a case for the notes of which I am indebted to Dr. Samuel Fenwick.

Case 2.—A gardener, forty-six years of age, was admitted into hospital in 1888, suffering from chronic dilatation of the stomach. It appeared from his history that he had always enjoyed excellent health until the age of forty-two, when he began to suffer from pain in the epigastrium after food, which was sometimes followed by vomiting. For three years these symptoms became gradually aggravated until he was unable to swallow any form of solid food without severe pain, and was accustomed to vomit several pints of sour fluid once or twice a week. But the immediate cause of his seeking medical advice was his intense dread of experiencing a return of what he termed "the convulsion spasms." It seemed that during the recent cold weather he had been two or three times attacked by sudden and severe vomiting, and almost imme-

diately afterwards the hands and feet had become stiff and painful, and he had suffered from cramps in the calves of the legs. He stated that the hands were clenched with the thumb inside, the feet drawn down, and the toes bent toward the sole. The spasms lasted several hours on each occasion, and left the limbs sore. During the attacks he could not walk.

On examination the patient was found to be extremely thin and anæmic. The heart-sounds were weak but clear, and the lungs presented no signs of disease. The stomach was greatly dilated, and its lower border extended to a spot three inches below the umbilicus, and a loud splash was audible on manipulation, while pressure over the pyloric region caused pain. There was no tumour to be felt. The bowels were obstinately confined, and the temperature of the body was slightly below the normal point. The urine contained neither albumen nor sugar. Partial stenosis of the pylorus, due to the contraction of a chronic ulcer in its vicinity, was diagnosed, and the stomach was accordingly emptied by means of a soft tube, and the organ thoroughly washed out. Under this method of treatment, combined with careful dieting, the patient rapidly improved, and finally left the hospital after having learnt to perform lavage for himself. In May, 1892, he again made his appearance. He stated that since his discharge from the hospital he had continued to use the stomach-tube several times a week, with the result that the pain and vomiting had been less severe than formerly, and the spasms had never troubled him again. The stomach was still dilated, but the patient had gained several pounds in weight. For the last two years there has been no news of him.

Although the notes of this case are very incomplete, inasmuch as the nervous symptoms were not observed by a medical man, there can be little doubt that the so-called spasms were in reality closely akin to those previously described, a recurrence of which it is probable the active treatment by lavage had tended to prevent.

In seven out of the sixteen cases the condition of simple tetany was complicated by the occurrence of convulsive attacks affecting the muscles of the neck, jaw, back, and face. These seizures took the form of intermittent spasms which lasted from a few minutes to half an hour, and generally left the muscles in a state of semi-rigidity. During

their continuance the patient was unable either to open the mouth or to swallow, and in several instances opisthotonos was a well-marked symptom. This variety of convulsion, which so often made its appearance in these cases, must consequently be considered as a form of tetanus; and, like the surgical variety of that disease, its appearance was always a sign of the deadliest import, for in every one of the seven instances death ensued from failure of the respiration.

In three cases out of the entire number the condition of tetany was said to have been associated with convulsions which were indistinguishable in their clinical features from those of ordinary epilepsy. The fits were usually repeated in rapid succession, and two out of the three cases proved fatal.

From these facts it would appear that, in a case of tetany associated with chronic dilatation of the stomach, the supervention of delirium, a rapid rise of temperature, and coma with dilated pupils are signs of serious import; while in those cases where spasms of a tetanic or epileptiform nature manifest themselves the prognosis is exceedingly grave.

Ætiology.—Three theories have been advanced to explain the occurrence of these convulsions in cases of dilated stomach.

1. Kussmaul supposed that the severe vomiting which usually preceded the onset of the nervous phenomena so diminished the quantity of fluid in the circulation as to render the nervous and contractile tissues abnormally dry; and that in this condition of excessive irritability any slight stimulus was sufficient to excite reflex spasm. But in the cases in question there is never any evidence of such severe depletion of the circulatory system; on the contrary, the pulse remains full and of good tension, and the patient never exhibits the withered and shrunken aspect of a dehydrated individual. It may also be noticed that in two of the recorded instances the initial vomiting was entirely absent. Of late years Kussmaul himself has expressed the opinion that the dehydration theory is quite untenable.

2. It has been suggested that the muscular rigidity is brought about in a reflex manner by irritation of the nerve endings of the pneumogastric in the mucous membrane of the stomach. But here, again, no satisfactory evidence has been adduced in support of such an origin of the disease, nor has the introduction of the gastric contents from these cases into

the stomachs of inferior animals been followed by any phenomena of interest.

3. The most plausible supposition appears to be that in such cases an organic poison is manufactured in the stomach, which by its subsequent absorption into the general circulation acts upon the various nervous centres as a convulsant. It is known that in the case of tetanus the poison which is responsible for the convulsions is absorbed from the site of its local manufacture, and it is therefore not unreasonable to suppose that a stomach whose contents is literally teeming with putrefactive organisms might occasionally prove a convenient receptacle for the development of a specific bacillus.

Another point in support of this theory is the well-known fact that in cases of poisoning by certain convulsants, such as strychnine, a spasm can often be induced by slight forms of peripheral irritation; and it has already been noticed that in these cases of tetany the mere percussion of the skin over the epigastrium or the administration of an enema was sufficient to induce an attack.

More recently Bouveret and Devic have drawn attention to the fact that many of the recorded cases exhibited a hypersecretion of hydrochloric acid, and they state that they have been able to isolate a substance from the contents of the stomach, closely allied in its chemical reactions to syntonin, which is capable of producing general convulsions when injected into the circulation of various animals.

Treatment.—Whatever may be the exact pathology of the disease, there can be no doubt that the main indication for treatment lies in the systematic use of the stomach-tube. It is true that in several of the recorded cases the passage of the tube appeared to excite an attack of tetany, but when this method of treatment was persevered with the result was usually a happy one, and in several instances, as in the second which I have cited at length, the convulsions never reappeared after the organ was once thoroughly cleansed and maintained in a comparatively aseptic state. Warm water is usually sufficient for this purpose, but if it is considered necessary to employ some antiseptic, resorcin or naphthol will be found to answer best.

	Author.	Age.	Sex.	Duration of gastric disease.	Nature of nervous symptoms.	Result.	Autopsy.
1	Kussmaul (*Deut. Archiv f. klin. Med.*, vol. vi, 1869)	27	F.	4 years	Tonic contraction of muscles of extremities. Attacks recurred several times, and lasted about 1 hour	Recovered.	—
2	Kussmaul (ibid.)	27	M.	14 years	Tonic contractions of muscles of hands and legs; general tetanic condition affecting muscles of back and neck	Death (coma)	?
3	Kussmaul (ibid.)	37	M.	?	Several attacks of tetany, affecting the hands, forearms, and legs	Death (coma)	Stomach dilated from contraction of pyloric orifice by chronic ulcer.
4	Neumann (*Deutsche Klinik*, 1857)	43	M.	Several years	Attacks of tetany lasting several days, followed by epileptic seizures for 12 hours	Death (coma)	Stomach dilated from contraction of ulcer near pyloric orifice.
5	Gassner (*Thèse de Strassburg*, 1878)	26	F.	Several years	Two attacks of tonic contraction of muscles of extremities, and two seizures of an epileptiform nature	Recovered.	—
6	Leven (*Maladies de l'Estomac*, Paris, 1879)	31	F.	Several years	Two attacks of tetany.	Death	Stomach dilated; chronic ulcer near pyloric orifice.
7	Leven (ibid.)	39	M.	11 years	Three attacks of tetany; interval of 10 days between first and second, 3 days between second and third; attacks lasted about 2½ hours	Death (coma)	Stomach dilated from chronic ulcer near pylorus.
8	Dujardin-Beaumetz (*L'Union médicale*, vol. xxxviii, 1884)	46	M.	5 years	Three attacks of tetany, associated with opisthotonos	Death	Stomach dilated from cicatrising duodenal ulcer.

9	Hanot (*Laprèvotte Thèse de Paris*, 1880)	56	F.	3 months	Three attacks of tonic contraction of muscles of hands and legs	Recovered	—
10	Dreifus-Brissac (*Gaz. Hebdom.*, No. 27, 1885)	32	M.	1 year	Three attacks of tetany of extremities, associated with spasms in muscles of back, jaw, and throat; attacks lasted 2 to 6 hours	Death	—
11	Nason (*Lancet*, ii, 1891, p. 44)	?	M.	Some time	Spasm of extremities and rigidity of neck and face	Recovered	—
12	Hayem et Gaillard (*Gaz. des Hôpitaux*, 1883, p. 900)	31	M.	?	Two attacks of tetany in extremities, first attack lasted 3 days	Recovered	Died two years later of cholera; stomach very dilated from stenosis of pylorus due to a chronic ulcer.
13	Martin (*Lancet*, Jan. 8, 1887)	48	M.	7 years	Only one attack of tetany, which lasted 4 hours	Death (coma)	—
14	Collier (*Lancet*, i, 1891, p. 1251)	48	M.	Several years	Tetany of arms with rigidity of muscles of back and jaws	Death (coma)	Dilatation of stomach; contracting ulcer near pylorus.
15	Paliard (*Revue de Médecine*, May, 1889)	43	M.	12 years	Tetany of extremities with slight remissions for 6 hours	Recovered	(History of hæmatemesis).
16	Mueller (*Charité Annalen*, xiii, 1889, p. 309)	44	F.	9 years	Tetany of extremities with epileptiform seizures	Death	Dilated stomach; chronic ulcer near pylorus.
17	Mueller (ibid.)	47	F.	13 years	Tetany of extremities with spasms of muscles of neck, throat, face, and of diaphragm	Death	Dilated stomach; chronic ulcer near pylorus.
18	Gerhardt (*Berl. klin. Woch.*, 1888, p. 74)	—	M.	?	Tetany of extremities; spasm of face; opisthotonos	Death (coma)	?
19	Renvers (*Berl. klin. Woch.*, 1884, p. 74)	—	M.	?	Tetany of extremities.	Death	Chronic ulcer of duodenum contracting pyloric orifice; dilated stomach.

	Author.	Age.	Sex.	Duration of gastric disease.	Nature of nervous symptoms.	Result.	Autopsy.
20	De Baurmann (*Soc. Méd. des Hôpitaux de Paris*, March, 1889)	33	M.	Several years	Tetany of extremities for 13 hours	Death (coma)	Dilatation of stomach from atony.
21	Merlin (*La Loire médicale*, Nov., 1890)	42	M.	Several years	Two attacks of tetany, with interval of 2 months; second attack proved fatal in 6 hours	Death (coma)	?
22	Loeb (*Deut. Archiv f. klin. Med.*, 46, i, 1889, p. 95)	64	M.	8 years	Two attacks of tetany each lasting 8 to 10 hours; sudden and fatal attack of general tetanus	Death	Stomach dilated from cicatrising ulcer near pylorus.
23	Bouveret et Devic (*Revue de Méd.*, Feb., 1892)	66	F.	1 year	Three attacks of tetany in 48 hours; each attack lasted about 3 hours	Death (coma)	Stomach dilated; chronic ulcer near pylorus.
24	Bouveret et Devic (ibid.)	33	M.	Several years	Three attacks of tetany	Recovered	—
25	Bouveret et Devic (ibid.)	37	F.	6 months	Tetany for ½ hour	Death (from gastric disease)	Cancer of pylorus and chronic ulcer of stomach.
26	Thomson (*Ann. Univ. Med. Sciences* (Sajous), 1888, p. 381	45	M.	Some time	Tetany of arms and hands	Recovered	—

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DESCRIPTION OF PLATE,

To illustrate Mr. F. S. Eve's Case of Tubercular Lymphangitis of Forearm and Arm following Inoculation of Index Finger.

Several tuberculous abscesses covered with thin livid skin follow the course of the lymphatics of the forearm and inner side of the arm. At the base of the index finger is a superficial ulceration.

V.—*A Case of Tubercular Lymphangitis following inoculation (?) of finger.* By Frederic Eve. *Read October* 12, 1894.

Elizabeth F., æt. 47, was seen by me as an out-patient at the London Hospital on February 14, 1890. The disease commenced by what she termed a whitlow on the left index finger. About two months before she came under observation a sore appeared on the dorsum of the finger, which healed and then broke down again. Five or six weeks later the arm became painful, red, and several swellings made their appearance.

Condition when first seen.—On the dorsum of the left index finger just above the distal joint was a superficial sinus, surrounded by a small superficial ulceration covered with flabby granulations. The sinus did not communicate with diseased bone. At the base of the finger over the metacarpo-phalangeal joint was a superficial cold abscess. Three similar prominent fluctuating swellings, covered with thinned and livid skin, were distributed in linear series along the dorsum of the forearm, and possibly corresponded to the valves on the lymphatics. On the inner surface of the lower third of the upper arm, and near the flexure of the elbow-joint, were several firm nodules the size of haricot beans, and some large and softer nodules. The firmer masses appeared to be tubercular formations around the lymphatics which had not yet suppurated. In the axilla were some much enlarged lymphatic glands.

She could not remember to have injured or wounded the index finger; and no consumptive had resided in the same house. She had never spat blood, and there was no evidence of phthisis or other tubercular lesion.

A diagnosis of primary tuberculosis of finger with tubercular lymphangitis was made. It appeared probable that the original trouble was a whitlow, the wound resulting from which was accidentally inoculated with tubercle,—perhaps, as was suggested to me, in washing a pocket handkerchief soiled with tubercular sputum. There was no diseased bone in the finger.

For the purpose of establishing the diagnosis some pus from one of the abscesses was drawn into a freshly prepared pipette, and a guinea-pig was inoculated beneath the skin of the groin. It died some weeks afterwards with well-marked general tuberculosis.

The patient was subsequently admitted to the hospital under the care of one of my senior colleagues, and the larger abscesses were opened. On March 10 the exuberant granulation tissue from the cavities of the abscesses was removed by scraping.

March 28.—Patient discharged with the wounds nearly healed.

She returned to me a year afterwards, in February, 1891, with a fresh crop of similar abscesses on the dorsum of the forearm, and one on the upper arm. The finger and the scars of former abscesses on the arm were soundly healed, and no enlarged glands were felt. Her general health continued good. She declined to have any further operation performed.

In May the abscesses were decidedly smaller, and after that she disappeared.

As I desired to watch the progress of the case I sought her out again in January, 1893.

She was readmitted with a sinus over the dorsum of the arm extending deeply between the muscles, but not connected with diseased bone. There were also some fresh ulcers on the hand and forearm; these were scraped. Her lungs were kindly examined by my colleague, Dr. Kidd, who reported, "I can only find a few scattered rhonchi. Beyond some slight bronchitis there are no abnormal signs in the chest." I have now lost sight of her.

Remarks.—An excellent account of the disease described above is given by Lejar in the 'Traité de Chirurgie' of Duplay and Reclus, published in 1890; a cast of an arm is figured precisely similar to the accompanying drawing. It was taken from a case of Bazin's in 1870, but the nature of the disease was not recognised at the time. The lymphangitis originated from an ulceration at the base of the thumb.

In a similar case described by Tuffier, a patient with a chronic ulceration of a finger was attacked by a series of tubercular abscesses following the course of the lymphatics of the upper extremity. Tuberculous disease of the glands of the axilla followed, and the patient succumbed to general tuberculosis.

It is worthy of remark, that in tuberculous disease of the glands of the neck and other similar affections, any lesion in the course of the lymphatic vessels is so rarely observed, although necessarily the tubercular virus must pass along them to the glands, and from one set of glands to another,—for example, from the neck to the axilla.

The reason that tubercular abscesses form in the course of the lymphatics in such cases as that related may be twofold. In the first place, and of greatest importance, is the fact that the tubercular virus is mixed with septic cocci and their products, derived from the primary lesion, which is usually ulcerated and suppurating; and in the second place the lymphatic channels passed before glands are reached are much longer, and the movements of fluid in them presumably slow, allowing the irritating virus to remain longer in contact with their walls. The presence of septic cocci must be granted, as it is well known that the inoculation of recent cultivations of tubercle bacilli is not followed by suppuration, although the subcutaneous injection of old cultivations is pyogenic.

As Lejar points out, in the published cases of tubercular lymphangitis, the disease nearly always succeeded the cutaneous inoculation of tubercle. In one case it is an anatomical tubercle, in another a warty tuberculosis on the back of the hand, and in three cases the patients were women who developed nodules of cutaneous tuberculosis on the fingers while nursing their phthisical husbands. Lymphangitis rarely follows deep tubercular lesions, as of the bones.

Goupil * has recently described four types of tubercular lymphangitis:

1. Lymphangite polynodulaire en série.
2. „ lymphangiectasique.
3. „ paucinodulaire à distance.
4. „ reticulaire.

Some of these sub-varieties seem hardly to merit a separate name, but the second form appears certainly distinctive.

One case has been recorded by Hallopeau and Goupil in a man aged sixty-nine, who exhibited an enormous swelling of the lower third of the left leg and instep. The skin was œdematous, like elephantiasis, and from this base a series of ovoid, red, small eminences of more or less firm consistence projected. These were due to enlarged and dilated lymphatics, some of which were intact and filled with

* Verneuil's *Études sur la Tuberculose*, tome iii, fasc. 2.

clear lymph, while others were open and ulcerated with lymph issuing from them.

Tubercle bacilli were found in large numbers in the lymph, and a guinea-pig inoculated with it died in two months from tuberculosis.

The primary disease was a tuberculosis of the tibio-tarsal articulation.

Treatment.—The importance of the early recognition of this disease, and of the adoption of prompt and energetic treatment, are obvious.

The abscesses or nodules should be laid open, thoroughly scraped, and treated with iodoform; and the nearest chain of lymphatic glands, if enlarged, should be excised.

The completed histories of several recorded cases tell that the patient ultimately died of consumption. The disease being at first distinctly local, the path of the virus towards the general circulation should be broken at the lymphatic glands.

VI.—*A case of Subdiaphragmatic Abscess communicating with the right pleura, successfully treated by resection of rib and drainage.* By Hector W. G. Mackenzie, M.D., and F. C. Abbott, M.S. *Read October* 26, 1894.

OLIVER H., æt. 10, was admitted to St. Thomas's Hospital on July 27, 1893. He was extremely ill, very weak, and much emaciated. He complained of pain and swelling in the abdomen. On examination there was found to be a hemispherical reddened acutely tender swelling centrally situated in the epigastrium. The right side of the chest in front was flattened, and moved little with respiration, while the movements of the left side were obviously exaggerated. In the recumbent position the percussion note over the right front was tympanitic except at the apex, where there was impairment of resonance. The same tympanitic note was obtained for about an inch and a half below the costal margin and over the epigastric swelling already referred to. Below this level the note became dull, and continued so to the level of the umbilicus, where the edge of the liver could be felt slightly descending with respiration. In the right axillary region the note was dull all over.

On auscultation the breath sounds were found to be absent or extremely feeble over the whole of the right side in front except at the apex, where there was loud bronchial breathing, with increased voice sounds and pectoriloquy. When the child was made to sit up, the front of the chest became quite dull all over, as also did the resonant area below the ribs. At the same time the upper part of the axillary region became resonant.

Behind, the note was dull all over. At the upper part the breath sounds were loudly and harshly tubular, vocal fremitus and resonance were increased, and pectoriloquy was very marked. Over the lower two thirds the breath sounds, vocal fremitus, and resonance were feeble or absent.

The heart was slightly displaced upwards and outwards, the apex being in the fourth space just internal to the nipple.

The note over the front of left lung was good, and the breath sounds were of an exaggerated puerile character.

Posteriorly the note was impaired towards the base, and there were numerous crepitations to be heard on auscultation.

To the above account it may be added that in front over the tympanitic area the *bruit d'airain* was well heard, that the abdominal swelling became very distinctly more prominent when the patient sat up or coughed, and that fluctuation could then be felt over it.

From the physical signs it was obvious that we had to deal with a pyopneumothorax communicating through the diaphragm with the upper part of the abdominal cavity, which was the seat of a subdiaphragmatic abscess. Moreover the abscess had undermined the skin, and was pointing in the situation of the swelling where the skin was red and thin.

The history of the illness was as follows. One day, about four weeks before his admission, the patient had indulged rather freely in cherries and apples. The same evening he complained, as was not unnatural, of a slight pain in his stomach. Next morning he woke with severe abdominal pain, and vomited a quantity of reddish-coloured matter mixed with what looked like yellow phlegm. He continued to suffer great pain, the seat of which at first was in the epigastrium and right hypochondrium. Subsequently pain was also felt in the right shoulder. Seven days after the onset of the illness the epigastric swelling was noticed. From the first he was not allowed solid food. Diarrhœa had troubled him for about ten days before coming to the hospital, the bowels acting about three times a day. He had had a cough for about three days; the temperature was not raised, although at first he was said to have been feverish. Previous to this illness he had been perfectly well.

What was the cause and nature of his illness? The history clearly pointed to an abdominal origin. First, there had been a subdiaphragmatic abscess, and subsequently this had perforated the right side of the diaphragm. The presence of air as well as fluid pointed to the perforation of an air-containing viscus. Although nothing in the previous history suggested the existence of a gastric or duodenal ulcer, still the perforation of such an ulcer seemed much the most likely cause of the phenomena which the case presented. It is not by any means uncommon to find in cases of gastric or duodenal ulcer, in which death occurs from hæmorrhage or perforation, that the characteristic symptoms of the disease have been absent.

The next question was one of treatment.

From the physical signs it was evident that the communication between the subdiaphragmatic abscess and the empyema was a very free one, and it was therefore decided to drain both through an opening in the pleura.

On the second day after admission a portion of the sixth rib was resected just behind the mid-axillary line. The pleura, which was scarcely thickened, was then opened, and over thirty ounces of exceedingly offensive, thin, watery pus containing some flakes were evacuated. Nothing in the nature of stomach contents could be detected in this fluid. The lung at once expanded well, the visceral pleura did not feel thickened, and the cavity promised to close well. On exploring with the finger the upper surface of the liver was felt through an opening in the fore-part of the diaphragm extending from about the tip of ninth costal cartilage nearly to the mid-line. The diaphragm and liver were partially adherent, and the exact limits of the subdiaphragmatic cavity could not be determined.

The whole cavity was well irrigated with warm boracic lotion, and a double drainage-tube inserted. This did not pass into the abdomen. The cavity was washed out daily with warm boracic lotion, and twice with a weak solution of perchloride of mercury. Two days after operation the edge of the liver was noted as being one inch above the umbilicus; the discharge was still very offensive, and the temperature 101·8°. Breath very offensive. Three days later the discharge was nearly sweet, and the general condition much improved.

On the thirty-fifth day the wound was almost healed, and there was no discharge. The right chest was resonant down to the wound, and the vocal resonance and fremitus were normal.

The wound was sound when discharged on the forty-seventh day. When seen six months later the boy had been perfectly well since his discharge; the wound was sound, and the lung fully expanded, the only sign being slight relative dulness on the affected side. Abdomen perfectly normal.

The surgical treatment in this case was merely that of an ordinary empyema, and the subdiaphragmatic abscess drained excellently by this route, better than it could possibly have done through an incision directly over it. The abscess was evidently above the liver, but whether originally of the greater sac it is impossible to determine.

VII.—*A Case of Multiple Neuritis, fatal on the fifth day.* By T. CHURTON, M.D. *Read October* 26, 1894.

IT is possible that the case about to be described will be regarded as one of Landry's paralysis rather than as multiple neuritis; but as Landry's paralysis is a very rare disease, while multiple neuritis is not a very common cause of death, and it is still less a common thing that death should be caused by it within a week, this case, in which the disease was fatal on the sixth day after the first observed symptom, may perhaps be, although imperfectly, put on record.

Miss ——, æt. 14, was seen in consultation with Dr. Martin (acting for Dr. Swann), of Batley, on July 31, 1894. The following notes of the case were written on the same day:—A tall, fair, very slender, undeveloped girl, looking younger than fourteen in the face. She had lost the use of her arms, legs, and trunk; there was absolute inability to turn in bed. No marked pain. Bones and joints normal. Twisting the ankle-joints causes pain, but this appears to be in the peronei muscles, and to result from tension produced in them by twisting the foot inwards. These muscles (peronei) and also the extensors of the fingers are tender; calf muscles are doubtfully tender. No reflexes anywhere—plantar, patellar, abdominal, epigastric, all absent. Even the eyelashes and conjunctivæ can be freely stimulated without any reflex movement of lids resulting, though she draws the head away to escape the somewhat disagreeable sensation. She can cross either leg over the other, but does so with a wide-ranging movement (imperfect co-ordination). Very little power in calf muscles; knee-raising power remains, but is small. No grasping power; can flex fingers slightly, has lost power of extending them almost wholly. Can flex and extend elbows, and can move shoulder-joints—all very feebly. Arms and legs about equally feeble. She cannot raise her head from the pillow; the power of using the muscles of neck and trunk is nearly gone; it is difficult to turn her over in bed, or to help her to attempt to sit up in bed. *Chest.*—The chest expands in breathing; the abdomen also moves (diaphragm). At times these movements are rapid, about forty in the minute. *Brain.*—Her replies, though rather

slow and languid, are normal; she is interested in her state, and though evidently tired by the examination, does her best to assist us in it. *Cranial nerves.*—Smell said to be normal. Vision seems unaltered; discs not examined. There are occasional spasmodic movements of the eyeballs; they travel conjugately towards the left. The eyelids sometimes also twitch; these movements have existed for a year. No squint; pupils equal. There is said to be a difficulty in mastication; it was not tested; the fifth nerve sensibility is normal. No choreic movements of facial muscles; no paralysis of them was observed. Hearing normal. There was said to be some difficulty in swallowing. A sense of "choking" accompanied the tachypnœa already mentioned. The heart's action was rapid—tachycardia. Sterno-mastoids very feeble. The tongue was protruded fairly well. There was a marked clipping of her words; in trying to count ten she only got as far as four—partly, perhaps, from the shortness of breath. No loss of sensation was anywhere found; a complete search, however, could not be made. On left buttock, near sacrum and coccyx, were two groups of wide bright red or crimson bands, not accounted for. No hyper- nor paræsthesia found. Sphincters normal; no difficulty in micturition and defæcation. She has never menstruated; the usual marks of sexual development are entirely absent. She has vomited this morning; it was supposed that a dose of sodium salicylate caused this. Bowels open by medicine. Nothing abnormal in abdomen. Neither sugar indican nor lithates in the urine. Lips rather pale. No enlarged glands found. Spleen? (The only note is "No swelling, &c., of abdomen.") There is a doubtful dulness of the base of right lung; vocal resonance is very distinct there; no pain; no marked cough; drew a deep breath twice fairly well upon being asked to do so. Heart-beat is quick; no bruit. No marked pulsation of carotids, &c. No œdema observed anywhere. Urine acid, sp. gr. 1032, no albumen. No perspirations; no rash on skin.

Causation.—She was always fragile. Her parents are alive and seem well, though they might perhaps be obscurely described as nervous. Patient is their third child; the second is quite well, but the fourth died of bronchitis after whooping-cough at the age of thirteen months. *Two years ago* they came to their present home. *One year ago* patient began to have twitchings of the eye or face—it was supposed from imitating a school friend. *A month ago* the twitchings

in eyes and face became worse, the shoulders became affected, and there was nodding of the head, &c. It is not known that she was at all feverish at this time. *Five days ago* (*i. e.* on Thursday, July 26) she went to a school party in the evening, was rather excited while there, and felt tired when it was over. She then missed the train for home, and was obliged to walk two miles; she felt exhausted on arrival. Next day (Friday) she felt tired all the day, had "pins and needles" in her fingers, and was unable to do her exercises on the piano. On Saturday she walked badly—staggering. On Sunday she kept her bed, complained of pain in the calves of her legs, with slight pain in the ankles. On Monday Dr. Martin found the temperature 103° and advancing paralysis. On Tuesday, when I saw her about 3 P.M., the paralysis of limbs was nearly complete.

In the evening of the same day the friends thought she was "choking." Next morning the breathing was quick and laboured, 40; pulse very feeble, 150; at 1.20 P.M. she died quietly, not struggling for breath, so far as could be ascertained.

Analysis of the water supplied to the house found ·07 grains of lead per gallon in the cold water, and ·308 grains per gallon in the hot water circulating through the house. It was learned that tea drunk by the patient had frequently been made with hot water taken from these pipes. The chain of causation would, therefore, appear to be constructed thus:—Constitutional debility; slight but constant presence of lead in the blood; exhausting effort; and some poison unknown.

That lead can cause or predispose to multiple neuritis is admitted; it happens to be the very first of the toxic causes of the disease mentioned in Dr. Gowers' classification (*Diseases of the Spinal Cord*, p. 111, 1892). If ever its power in this respect should be doubted, I believe the West Riding of Yorkshire could furnish abundant proof in its favour, and new fields of study of the subject are being constantly supplied to us as efforts at the more complete supply of the resources of civilisation to our towns and villages are being more energetically made. That the disease in the present instance was multiple neuritis may be more doubtful; but neither pure Landry's paralysis nor pure anterior polio-myelitis would have begun with "pins and needles" in the fingers, nor subsequently would either of them have caused the pain in the calves of the legs and the ankles, nor, later, tenderness in the peronei muscles and the long extensors in the forearm. The order of

invasion, again, is not that of acute ascending paralysis: ataxy was noticed first in the hands, next day her gait was staggering; only on the *third* day she was in bed.

But the questions beyond that of exact diagnosis one would desire to be allowed to ask with respect to the case are (1) whether any treatment has been found of any use in such apparently desperate circumstances, treatment resembling that of grain doses of ergotin hourly to the number of twenty, which seemed to cure a case of Landry's paralysis, as related by Dr. Gowers (op. cit., p. 386); (2) what share may fairly be attributed to lead in the causation.

VIII.—*A case of Poisoning by one ounce of Chloral Hydrate: recovery.* By R. J. Colenso, M.B. (Communicated by Sir Dyce Duckworth, M.D., LL.D.) *Read October* 26, 1894.

ON December 12, 1893, at 4 P.M., a single lady of gymnastic profession, æt. 34, was found by her mother in a state of profound coma. Medical aid was not at once procurable, owing to absence of the ordinary attendant. Until his arrival an hour and a half later, at 5.30 P.M., every domestic means of arousing the patient was vainly exhausted by her mother, who alone was with her during this trying interval.

Beyond subacute dyspepsia during the previous spring, and some anæmia, this lady had suffered from no serious or chronic complaint; she had, however, an excitable and restless disposition, undergoing in the exercise of her profession an excessive amount of great bodily exertion with a considerable degree of mental worry; in fact, she was physically and mentally over-strained. All functions were regular, catamenia rather profuse with some anæmia, for which cod-liver oil had been taken.

Her mother gave the following account:—At 8 A.M. complaining of a sleepless night and a severe headache, she had stated her intention of taking a dose of a mixture prescribed some weeks previously, and had given strict orders that she was on no account to be disturbed. As she did not appear at luncheon, from this hour (1 P.M.) attention was drawn to an absolute silence in her bedroom, a room above where her mother was sitting, and becoming more and more anxious, the mother at last essayed to waken her at 4 P.M., finding her totally unconscious.

The summons which was received more than an hour after this discovery gave me a bad quarter of an hour, for it was thus worded:—"Miss X. Y. took a dose of your medicine at 8 A.M. this morning, and has been insensible ever since."

The medicine thus referred to was found in a far corner of her room; an eight ounce partly-empty bottle, some two weeks old, of the following prescription:—Ammonii Bromidi gr. xv, Tinct. Nucis Vomicæ ℥vj, Sal Volatile ℥xx, to be taken occasionally in sufficient water.

A second eight-ounce bottle containing two ounces of a mixture was found on a small table by the bedside at her

elbow, and a glass stained with a few drops of fluid of a similar colour, taste, and odour. The prescription for the latter was belladonna tincture ℥vj, dilute hydrocyanic acid ℥iij, aromatic sulphuric acid ℥xx, compound infusion of gentian to one ounce, to be taken thrice daily.

Clearly, to neither of such prescriptions, rightly dispensed, could her state be attributed, nor did the drugs seem other than they purported to be; nevertheless, and particularly as the latter had been, contrary to advice, dispensed by a "store chemist," these were locked up for future reference, together with the washings of the stomach, and the urine first drawn off. Since, for some four months previously, in addition to hard physical work and excitement, the lady had been suffering mentally from an *affaire du cœur*, "*notumque furens quid femina possit*," I made a diligent search for some more satisfactory cause of her serious condition, suspicion being presently confirmed by the discovery in a screened and empty grate of the freshly torn and crumpled wrapper of some small bottle.

At 5.30 the patient's condition was as follows:—In supine decubitus, with deep coma and motionless, with face and conjunctivæ slightly congested; that of the left eye more so, and in this eye a normal slight external strabismus is more than usually pronounced. Conjunctival reflex and almost all others save that of deglutition, which is very imperfect and slow, abolished. Breathing very shallow, and occasionally stertorous. There is a slight odour only of brandy, which had been applied by her mother, and her jaws are tightly locked. The pupils are equally contracted to a third of normal size, and respond sluggishly to light. Pulse regular, 130, of small volume and rather high tension; temp. 100·5°. For the preceding hour her mother unaided had vainly exhausted all the usual household remedies in trying to arouse her, but met with no response beyond some slight twitching of the face.

A four-minim hypodermic injection of sulphate of atropine solution was at once given, it being thought more probable from the length of time the patient had endured the poison, and also from the pupillary contraction, that some form of opium had been taken. After this no time was lost in washing out the stomach with warm water, which returned almost unaltered. Here the want of a nasal tube was badly felt, for it was exceedingly difficult to unlock her jaws, a crown of one of the molars being chipped by the wedge used

for an old-fashioned two-way stomach-pump, the only available instrument, of which the tube was next bitten almost in two, and could not subsequently be used to feed her through.

In about ten minutes, her pupils being unaltered, a second similar dose of atropine was given. As deglutition was so imperfect, after several trials, feeding by the mouth, with small (ʒj) doses of a mixture of milk, Valentine beef essence and brandy was discontinued, hypodermic injections of ether ℥v being given every twenty minutes. Continual firm friction of the limbs, with occasional smart slapping, was also commenced at this time by three lady friends and a trained nurse, and was used continuously for ten hours. No appreciable result was obtained by the atropine, and in spite of this stimulation the coma became more profound, and the pulse often almost faded away, having now fallen to half of its original volume, with an unaltered frequency. When this occurred cautious inhalation of nitrite of amyl was used, and for a minute or two after use certainly improved the pulse, increasing its volume and slightly lowering its frequency. Liquor Strychninæ, 1 per cent. solution, also in three to five minim doses, was used hypodermically every twenty to thirty minutes, sometimes in conjunction with the ether, at other times alternately with it. At 7.30 urine was drawn off; it was of a dark sherry colour, and about ten ounces in quantity.

At 10 P.M. Sir Dyce Duckworth, arriving very promptly and kindly on a summons, approved of our treatment, and recommended in addition enemata of hot coffee, an application of mustard plasters to the legs and thighs, and hot water bottles to the trunk. The nature of the poison taken was until now a mystery, soon to be solved by a lady present, who confessed that the above-mentioned wrapper was one in which she had on the previous evening procured some chloral for the patient who desired it "for a friend in India." Its amount was said to be one and a half ounces of pure chloral hydrate in large crystals dissolved in sufficient water, forming a saturated solution of one grain to two minims. This had been with difficulty procured after a visit to several neighbouring druggists.

The vendor was at once summoned by Sir Dyce Duckworth, and before us, in the presence of this lady, made a clean breast of it, identifying his wrapper, and acknowledging to one and a half ounces of chloral dissolved in three ounces of water; Sir Dyce Duckworth thought badly of the

patient's chances of recovery, and during the time he was present there was a marked failure of the pulse. Shortly after he left, an enema of hot coffee, brandy, milk and Valentine beef essence was given and retained.

On smart slapping of the legs and forearms there was at 12.30 some twitching of the face, and in half an hour the limbs began to be drawn up on slapping. Her temperature was now 102° F.; the pulse 120, very small but less variable in volume. In an hour's time the patient began to groan and to throw herself about, and as she became very restless by 2 A.M. the hypodermic injections of ether and strychnine were discontinued, and the sinapisms intermitted for half an hour at a time, being applied only when she again seemed inclined to become comatose. Since this happened whenever she was left alone for a few minutes the massage was continued, although she now began to articulate, begging to be "left alone," to be allowed to "go to sleep." A second enema was given at 3 A.M., and the friction continued for another hour, at the end of which she swallowed some milk and brandy, and opening her eyes began to take notice, plainly recognising some of the bystanders.

She was next allowed to sleep in snatches of ten minutes at a time, slumber coming on almost instantly, and becoming in a few minutes semi-coma, yet when roused the patient was well awake and sensible. After an hour and a half of such treatment she was induced, on the promise of being allowed a good sleep, to get out of bed between two assistants, and supported and encouraged to move around the room; and this was achieved, although she was quite unable to stand alone, and kept her eyes closed during most of the time, a period purposely lengthened out by making her bed. At 5.30 A.M. she was again given some more nourishment, and put to bed with hot water bottles, and the nurse instructed to wake and feed her again in half an hour, and to send for assistance should the patient again become comatose. It was now twelve hours since she had been continuously under observation and treatment, the chloral having been latterly admitted by her to have been taken as early as 8.30 A.M. on the previous day, or eight hours earlier, making a total of twenty hours before she was safe from its immediate effect. Yet at 5.30 P.M., or seven hours after the poisonous dose, her general condition was far better than it was a few hours later, and the pulse showed rapid deterioration at 7.30 P.M. The nature and quantity of the poison has been established since her convalescence, during

which a discovery was made. It was noticeable that for a week or two she shrank from being left alone by her nurse, but was more composed after the removal of a suspected and locked cash-box. This box was an object of suspicion, because, on being moved, it evidently contained some loose glass; and being broken open, it was found to contain two medicine bottles, verified by the chemists from whom they were procured, viz.—

1. A stoppered three-ounce phial labelled "Solution of chloral one grain in each two minims of solution. Poison." From this exactly two fluid ounces, containing 480 grains of pure crystallised chloral hydrate, were missing, leaving one fluid ounce of this saturated solution, dispensed December 11, 1893.

2. Also a second smaller wide-mouthed six-drachm bottle, exactly containing and brimful of hydrate of chloral in large dry crystals, labelled "Crystal chloral, for insect bites only. Poison," which was dispensed in 1892.

This latter had been procured for mosquito bites for external application, and had been clearly kept intact for two years—an abstinence hardly possible to an habitual chloral eater, nor, indeed, during a three years' attendance had she shown any symptoms of such a habit.

Of the former bottle full of solution, said to have been procured for a friend in India, two thirds had been taken in a sudden frenzy, in a single dose, as the patient now admits, and in an ounce of some other mixture, but not that in which she believed she had taken it, the time of her doing so being uncertain; for although she thinks it was at 8.30, her memory is weak and confused. Until convicted by the discovery in her cash-box she positively denied all knowledge of her act, or of any intention of so doing, although naturally remarkably courageous and truthful, or of having taken other than a dose of non-poisonous mixture. Some eight weeks afterwards I wrote to her, and obtained the following replies.

1. When did it happen? "The dose must have been taken after going down to mother's room," *i. e.* 8 A.M.

2. When did you lose consciousness? "I have not the least idea when this happened."

3. Was it taken in one or more doses? "All at once, I think."

4. In what vehicle? "I believe I must have taken it in some of your medicine."

5. When did you recollect the act? "On my return

from Eastbourne, when mother told me of it," *i. e.* January 16, five weeks afterwards."

6. When did you regain memory? "Of the time, and until Thursday morning, I remember absolutely nothing, *i. e.* until December 14th, two days later."

Her subsequent history is as follows:—The bladder and bowels were naturally relieved on the next morning at 8.10 A.M. and 9 A.M. respectively, and a good deal of flatus passed *per vias naturales ambas.* The temperature, which reached 103° F. at the midnight of her seizure, gradually fell to slightly subnormal by the fourth day, December 15, but on the following day suddenly rose to 103·8° F., the reason for this being discovered in a burn of four square inches area on the outer side of her right thigh, overlooked by the nurse, and caused by a hot water bottle in conjunction with a wandering mustard plaster. This was dressed with lead lotion for a day or two, and healed up well and quickly with an application of subgallate of bismuth in powder.

During the first twenty-four hours from 8 A.M. on the following morning, December 13, she slept thirteen and a half hours, but for some nights subsequently and occasionally during her convalescence it was found necessary to exhibit sulphonal at bedtime. For many days the digestion was irregular and imperfect, and the liver torpid or obstructed. Muscular tone was for long subnormal, and she was unable to be downstairs until January 7, going out in a carriage on the 8th. Tone is still not quite regained, although she is back at her professional work. At intervals during the first six weeks of convalescence, and notably on her visit to Eastbourne in January, she had periods of great excitement alternating with equal depression, so that a nurse was considered indispensable.

Remarks.—It was not until after the hypodermic use of strychnine between 8 and 9 P.M. that there were any signs of reflex or other reaction; ether alone was insufficient, the total amount of strychnine administered being nine tenths of a grain. To Sir Dyce Duckworth's suggestion I owe the use of nutrient enemata, for so little food was swallowed or digested that other administration was practically useless. The plasters also were very useful in provoking reflex action, so much so that they had soon to be discontinued.

A bad burn caused by an overlooked plaster showed with even a trained nurse the necessity for minute medical supervision.

The temperature, as in some other recorded cases, rose to 103° during the first twenty-four hours, subsiding on the fourth day, with a subsequent rise, due only to a skin wound.

The amount of chloral taken is clearly established to be that of a solid ounce of hydrate of chloral in large pure crystals, dissolved in two fluid ounces of water. This exceeded the amount taken in Dr. Levenstein's case, the exact amount being rather hypothetical in the case contributed by Dr. Plummer to the *Lancet*, January 6, 1894. Whilst in the case of Professor Tyndall, who was physically weakened by ill-health, and perhaps by the shock of the *contretemps*, the quantity swallowed was an ounce; but part of this was rejected by the use of emetics, and his death was probably therefore due chiefly to the former causes.

REFERENCE MAY BE MADE TO THE FOLLOWING AUTHORITIES.

Maximum Dose.

1. LIEBREICH, *Brit. Med. Journ.*, April 15, 1871.
2. CURCHMANN, *Brit. Med. Journ.*, April 15, 1871.
3. RICHARDSON, "Lectures on Experimental and Practical Medicine," *Lancet*, February 11, 1871.
4. ANSTIE, *Practitioner*, 1871.
5. *Lancet*, May 13, 1871.
6. LAURANCE, *Lancet*, March 4, 1871.

Fatal Dose.

1. WATKINS, *Brit. Med. Journ.*, February 25, 1871.
2. MORRIS, J., *Lancet*, February, 1871.
3. LEVINSTEIN, *Lancet*, February 25, 1874.

Tolerance of the Drug.

BRUNTON, *Pharmacology, Therapeutics, and Materia Medica.*

Fatal Period.

GUY and FERRIER, *Forensic Medicine.*
CRICHTON BROWNE, *Lancet*, April 1, 1871.

IX.—*A case of Pulsatile Congenital Cervical Cyst simulating Aneurysm.* By C. H. Golding-Bird. *Read November* 23, 1894.

F. W. H., æt. 36, was admitted into Guy's Hospital under my care on March 13, 1894.

He stated that he often had had the glands in the neck enlarged after exposure to cold, he being a railway signalman; and when in September, 1893, he first noticed the present tumour as a small soft lump, he thought it also was a gland. In consequence he had several carious teeth removed without effect, and the tumour steadily increased up to the present time. It has never been inflamed or painful, and is always soft.

There is now in front of the sterno-mastoid, on the right side, in the superior carotid triangle, a soft, oval, well-defined, fluctuating swelling; over it the parts are freely moveable, but it appears deeply attached itself, and it extends from just below the angle of the jaw to the upper border of the thyroid cartilage. It is markedly pulsatile, and pressure on the carotid below arrests pulsation. The pulsation is most marked antero-posteriorly, but there is also a very clear but not so forcible lateral dilatation. There is no bruit. With the neck extended the tumour cannot be lifted off the artery so as to modify pulsation; but with the sterno-mastoid relaxed the tumour can be so far lifted up as very considerably to modify, though not completely remove, all pulsation. (Under an anæsthetic, with everything relaxed, the tumour could, however, be lifted from its bed, and pulsation thereby stopped.) The temporal pulses are synchronous and of equal volume.

The diagnosis was that of cyst—probably a breaking-down gland. I removed it by operation, and found that it lay beneath the deep fascia over the carotid triangle. It had no evident structural relations with the surrounding parts; and once exposed, mere tearing of the surrounding cellular tissue sufficed for its removal without injury.

An examination of it showed it to be an oblong, thin-walled, flaccid cyst, only partly distended with grumous oily contents. Held up by one pole, it was egg-shaped; laid on its side, it flattened out to twice its width, losing proportionately in thickness.

Microscopic examination of the wall, after due preparation, showed it to be that of a congenital sebaceous cyst.

Now all present are doubtless familiar with Holmes' masterly and exhaustive essay on pulsatile tumours, in the *St. George's Hospital Reports* (vol. vii), and it might well seem that nothing more remains to be said on the subject; but two reasons at least have prompted me to publish this case. In the first place, though many kinds of cysts—using this word in its widest sense—have been found, at some time or other, to afford instances of pulsatile tumours, I have not yet been successful in finding the report of a case where a congenital cyst has taken on itself the characters of an aneurysm; and, in the second place, this case gives an opportunity for discussing why some tumours should have pulsation communicated to them and some not, when both, as far as can be made out, bear identical relations to some large artery. To take this second point first, I would remark that the situation of this tumour was the one of all others in which, perhaps, most cysts are found. Nothing is more frequent than to have to deal with tubercular abscesses anterior to the sterno-mastoid and deeply situated,—indeed, so deeply as by no means infrequently to be more or less adherent to the internal jugular vein; and yet it has but once fallen to my lot, that I can remember, to see one in which pulsation communicated by the carotid has raised the question of aneurysm. Clearly the mere proximity of a fluid sac to the wall of an artery, even when confined beneath such a firm covering as the extension of the deep cervical fascia anterior to the sterno-mastoid, will not alone explain it. Where, too, a certain degree of pulsation is communicated, it is almost always of the direct, forward, lifting kind, and unaccompanied by that lateral expansion which is such a distinguishing feature of a true aneurysm, or at least of a cyst cavity into which blood finds access, or of a soft solid tumour into which blood still in its vessels enters to such an extent as to confer upon it the characters almost of erectile tissue. And, speaking of such cysts and tumours, Holmes remarks* that "the lateral pulsation on which so much stress is justly laid in the diagnosis of aneurysm shows only that the fluid is contained in a large cyst, and that an artery opens freely into it. It (the lateral pulsation) cannot by itself show whether this cyst is purely arterial or is excavated in the substance of a tumour." Now in this case that I have

* *St. George's Hospital Reports*, vol. vii, p. 184.

narrated, in which neither of these conditions was present, lateral pulsation was marked, making the resemblance to aneurysm the more real; and a study of the tumour after removal, to my mind, gives the key to this unusual and puzzling symptom.

The cyst as I have mentioned, was thin-walled and flaccid, though filled with—but not distended by—an oily contents. When laid on the palm of the hand it did not present the egg-shaped form that it had when held up by one extremity, but became flattened, and on the slightest agitation it—if I may be permitted a very significant schoolboy's expression—"wobbled" from side to side. The flaccidity of the cyst explains this, and accounts also for the fact—and this is the key we are searching for—that when laid across a finger it *wrapped round it.* Now picture such a tumour in the anterior triangle of the neck, with its congeries of various arteries, and held down beneath the deep cervical fascia; and, further, the fact that its loose character allowed it to partly wrap itself round a large arterial trunk; and we have at once all the conditions but one that would furnish the objective signs of aneurysm; and yet the tumour lacked any communication with the interior of an artery. The pulsation of the artery would be directly transmitted in the anterior or lifting sense, the cyst being pressed on it by the deep cervical fascia; and the lateral pulsation would be caused by the fact that to all intents and purposes the artery ran through the tumour, or, what was the actual fact, the thin-walled flaccid tumour wrapped itself round the artery. Had the cyst been inflammatory in origin, the stiffness of its walls and its adhesion to surrounding parts would have prevented its applying itself thus closely to the artery; and this fact may explain why, in deep cervical abscesses, pulsation is not more frequently met with than it is.

An abscess may, however, present all the characters—save that of bruit—of an aneurysm, even to marked lateral pulsation, if large and flaccid enough, and if pressed firmly on the vessel by some inelastic covering, and if the vessel have a sufficiently powerful pulsation itself. Such a case I published in 1883,* where a large abscess in the anterior mediastinum rose above the sternal notch, and presented all the physical signs of an aneurysm save bruit; and the conditions for these were rendered evident after the abscess had been opened, since the finger could be then passed down behind

* *Guy's Hospital Reports*, 1883, p. 83.

the sternum into a cavity into which the arch of the aorta prominently projected. In this, as in the case related to-night, a bruit was absent, the condition for the production of this symptom being present in neither case. Nor was there serious difficulty in coming to a correct opinion in either case.

As to the former of the two points which I mentioned at the commencement of my remarks—the nature of the tumour,—this was set at rest by a microscopic examination of its walls, which showed it to be a congenital cyst, a tumour which, as I said, I have not met with before as simulating aneurysm; and, lastly, this case would seem to teach that a cyst structurally unconnected with a blood-vessel must, in order to show lateral or expansile pulsation as well as that directly forward, be flaccid enough to apply itself so closely to an artery as more or less to embrace it; or that the artery must be sufficiently large and have a powerful enough beat to impress its form into a proportionately yielding or thin-walled cyst.

X.—*A case of Splenic Leuchæmia greatly improved by treatment with inhalations of oxygen and with arsenic.* By Frederick Taylor, M.D. *Read November* 23, 1894.

THE following case of leuchæmia seems to me deserving of record on account of the remarkable improvement, which the patient experienced in the course of two months' treatment, as regards both the amount of leucocytes and the size of the spleen. The notes are from the very full report of Mr. H. Cooper, my clinical assistant at the time. The patient was admitted under my care, but in accordance with the arrangements of our clinical wards he afterwards passed under the care of Dr. Pye-Smith, who continued the treatment on the same lines.

Joseph H., æt. 25, a policeman, was admitted into Guy's Hospital on November 2, 1892. He is a native of Westmoreland, and has lived in Shropshire and Yorkshire. The last three years he has lived in London. He has never been out of England. He has always been temperate. He has had scarlet fever, measles, and quinsy, gonorrhœa at twenty years of age, and influenza a year ago. Has never had ague or any malarial fever.

Family history.—His parents, four brothers, and four sisters are all alive and well. There is no consumption in his family. He is unmarried.

Present illness.—He first noticed about five months ago that his abdomen was hard all over, and he felt some weight in the abdomen at the same time; but he had no further inconvenience nor pain, and worked as usual. He has been losing his colour for about a year, and his flesh and strength for some time. Three weeks ago he had severe pain in the abdomen, which lasted two or three days, and was accompanied by diarrhœa. On October 23 he had sudden severe pain in the left lumbar region, which lasted then about two hours, and has troubled him from time to time since. It is aggravated by walking, and he cannot lie comfortably on the left side. From the first he has progressively lost colour, strength, and weight, until the last two months, during which he thinks he has increased a little in weight. He is

also tired, languid, and short of breath. His appetite is good; he has had no cough, and has not noticed any enlarged glands. He has had frequent attacks of epistaxis during the last five months. The divisional surgeon reports him to have been ill as follows:—May, 1891, influenza; November, 1891, rupia; August, 1892, catarrh, abdominal swelling; October, 1892, enlarged spleen.

Present condition.—A powerful-looking man, of fair complexion and hair, with a faint pink tinge on the cheeks. Height 5 feet 10 inches. Weight 11 st. 9½ lbs. Abdomen full, prominent, rounded, and tense; rather more prominent on the left than on the right side. The umbilicus is flattened out, and one or two veins are visible running down to Poupart's ligament. The left half of the abdomen is occupied by the spleen. It forms a smooth, hard, resistent tumour, which extends also into the right side, and presents a well-defined edge to the right, with a distinct notch at the upper part. The lower border of the mass is three inches below the umbilicus. Behind the anterior superior spine of the ilium an ill-defined edge can be felt, which becomes more definite in the flank; and this rounded posterior border is six inches from the spine of the third lumbar vertebra. The spleen passes above under the ribs, and its upper limit, as ascertained by percussion, is 4⅜ inches below the nipple in the nipple line. The tumour is moveable, painless, and not tender. A creaking rub can be heard over the greater part of the left side of the tumour at the end of inspiration. The measurements of the spleen are as follows:

Horizontally at level of umbilicus	12	inches.
Greatest transverse measurement (1⅜ inches above umbilicus)	13¼	,,
Oblique measurement in course of tenth rib	14⅝	,,
Vertical measurement through umbilicus	7⅝	,,
,, ,, in nipple line	10⅛	,,
,, ,, through anterior superior spine	8⅞	,,
Extreme limit to right of umbilicus (1½ inches above umbilicus)	2⅞	,,
Distance of upper border below tip of xiphoid cartilage in umbilical line	2⅞	,,

The hepatic dulness begins above at the fifth intercostal space, but does not extend below the ribs; nor can the liver be felt.

The cardiac impulse is in the fourth space, immediately below, and internal to, the nipple: there is no increase of the precordial dulness. There is a faint pulmonary systolic murmur.

The chest is flat, with rather everted costal margins, and a very wide epigastric angle. There is a creaking rub at the end of inspiration in the left lower axilla, continuous with that heard over the spleen: otherwise respiration is normal.

The urine is clear, lemon-coloured, acid, of specific gravity 1010, containing urea, 1·4 per cent., no albumen, no sugar, and no blood.

Blood.—He does not show extreme anæmia: there is a distinct though faint pink tinge on the cheeks; there is some pallor of the lips, gums, eyelids, and fingers, but it is not pronounced.

A specimen of the blood placed under the microscope shows a field covered with leucocytes, while here and there are rouleaux and clusters of red discs. The leucocytes appear to be as numerous as the red discs; they are large and granular: measure about 10 μ. Some of the leucocytes contain large oval granular nuclei; others have two or three nuclei. Some of the red discs appear nucleated. The number of corpuscles in a cubic millimetre is 2,880,000: of these 1,880,000 are red corpuscles, and 1,000,000 are leucocytes. Hæmoglobin appeared to be 40 per cent. of the normal. There is no apparent enlargement of any lymphatic glands, and no evidence of the enlargement of those in the chest or abdomen. Optic discs normal; retinal veins tortuous, but doubtful if more so than is natural.

He was ordered on November 3 to have 5 minims of Liquor Arsenicalis in solution three times a day, and to inhale 30 litres of oxygen gas daily.

The dose of Liquor Arsenicalis was increased to 7 minims on November 7, to 9 minims on November 11, to 11 minims on November 18, to 13 minims on November 21, and to 15 minims on November 25.

On the lower third of each leg are a few patches of pigment, occupying an area of 3 inches by 4 inches; they are of a dark brown colour, with irregular, somewhat scalloped outlines, not raised above the surrounding surfaces; here and there they show some thin silvery scales. They are suggestive of syphilis; but questioned on the subject, he denies any primary or secondary lesions. The "rupia" above mentioned was only a week old when he applied to

the surgeon. There were, in addition to the patches on the legs, a patch of dry scales on his left knee, and one on his right thigh. He took medicine for three weeks.

November 18: He was ordered one grain of Pilula Hydrargyri, ter die. November 19: Examination of blood. Red corpuscles per cubic millimetre, 3,080,000, or 61·6 per cent.; leucocytes, 470,000; hæmoglobin, 40 per cent. November 28: The oxygen inhalation has been continued, to the extent of about 30 litres in 24 hours. He feels better, and eats and sleeps well. The spleen has decreased somewhat in size, especially at the lower and right margins.

Horizontal measurement at level of umbilicus	10½	inches.
Greatest transverse measurement (2 inches above umbilicus)	14	,,
Vertical measurement through umbilicus	5	,,
,, ,, in nipple line	9¾	,,
Extension beyond umbilicus	2¼	,,
Distance of upper edge from point of xiphoid	2¾	,,
Distance of lower edge from symphysis pubis	3	,,

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December 2: He has been troubled with nausea, but as it is not severe the arsenical mixture has been persisted in.

December 5: The blood is remarkably different from what it was. A drop placed under the microscope does not show more than two or three leucocytes in the field, and did not, indeed, strike Mr. Cooper (the clinical assistant) as showing an abnormal number. Examination showed: red corpuscles, 3,090,000 (61·8 per cent.); leucocytes, 10,000; hæmoglobin, 35 per cent.

On December 8, as he was showing some indications of the toxic effects of arsenic, the medicine was discontinued for a time, and he took Tinctura Ferri Perchloridi, with a few grains of Sulphate of Magnesium. The mercurial pill was omitted.

On December 16 the measurements of the spleen showed a further diminution, as follows:

Transverse	11½ inches.
Vertical measurement through umbilicus	none
„ „ in nipple line .	8 „
„ „ at meeting of upper border with costal margin . .	7½ „
Extension beyond umbilicus. . .	none
Distance of upper border from xiphoid.	2¼ „
„ of lower border from symphysis pubis	4⅜ „

It may be noted that the umbilicus lies slightly to the right of the actual median line of the abdomen. December 18: Examination of the blood. An undiluted specimen under the microscope shows from 10 to 12 leucocytes in the field. Enumeration gives per cubic millimetre:—Red corpuscles, 3,630,000, or 72 per cent; leucocytes, 20,000; hæmoglobin, 40 per cent.

The patient left the hospital on January 4, 1893, seeming in pretty good health. The spleen was about the same size as on December 16.

Longest oblique measurement nearly in course of tenth rib	13 inches.
Transverse measurement at level of umbilicus.	5½ „
Transverse measurement at level of junction of upper border with costal margin	10½ „
Vertical measurement in nipple line .	8 „
Distance from umbilicus . . .	2 „
„ „ nipple	2½ „
„ „ symphysis pubis . .	4½ „
„ „ anterior superior spine of ilium	¾ inch.

The urine usually varied between 40 and 60 ounces per diem. The specific gravity was mostly between 1014 and 1018, and the reaction was always acid. The urea was estimated on twenty-three separate occasions, and in sixteen instances was 2 per cent. or more, mostly between 2 and 2·5. Estimated daily amounts were between 400 or 600 grains. Deposits of crystalline uric acid were frequently observed.

The record of the temperature has, unfortunately, been mislaid.

After leaving the hospital he went into Yorkshire, and we have heard, through the kindness of Dr. E. P. Pickersgill,

of Sherburn-in-Elmet, that on January 11, 1894, he was losing flesh, that there was still a cardiac bruit, and that the spleen was very much enlarged, reaching to Poupart's ligament, and extending right over to the right side of the abdomen. The blood was not examined.

Dr. Pickersgill again writes on August 14, 1894, that "the spleen is extremely enlarged, although he has been under Liquor Arsenicalis, ℥xv, thrice daily for some weeks now, with slight intermissions."

The following records of his weight have been given me:—On November 6, 1892, while in Guy's Hospital, 11 st. 9½ lb.; soon after leaving the hospital, 14 st. 4 lb.; in December, 1893, 13 st. 7 lb.; in August, 1894, 12 st. 11 lb.

In order to show at a glance the progress of the case when under our care, I have tabulated the observations on the blood and on the size of the spleen respectively.

Condition of the Blood.

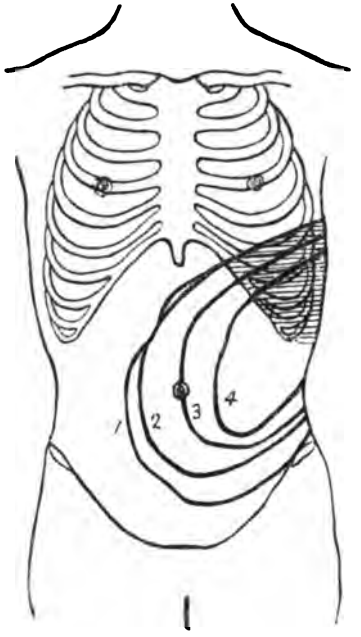
	On admission.	Nov. 19.	Dec. 5.	Dec. 18.
Red corpuscles per cub. mm.	2,880,000	3,080,000	3,090,000	3,630,000
Percentage of normal	57·6	61·6	61·8	72·6
Leucocytes per cub. mm.	1,000,000	470,000	10,000	20,000
Relation to normal (8000)	125 to 1	61 to 1	1·25 to 1	2·5 to 1
Hæmoglobin percentage of normal	40	40	35	40

Size of the Spleen.

	On admission. Inches.	Nov. 19. Inches.	Dec. 16. Inches.	Jan. 4. Inches.
Greatest transverse measurement	13½	14	11½	—
Oblique measurement	14⅝	—	—	13
Vertical measurement at umbilicus	7⅛	5	—	—
„ „ in nipple line	10½	9¾	8	8
„ „ opposite anterior superior spine of ilium	8⅞	—	—	—
Vertical measurement at line of junction of upper border with costal margin	—	—	7½	—
Extension beyond middle line, short of the middle line	2⅞	2½	—	2
Horizontal measurement at umbilicus	—	10½	—	5½
„ „ at junction of upper border with costal margin	—	—	—	10½
Distance of upper border from tip of xiphoid	2⅞	2⅝	2½	—
Distance of lower border from symphysis pubis	—	3	4⅛	4½

The figure on the opposite page represents the size of the spleen at different dates.

FIG. 1.



1. November 2, 1892.
2. November 19, 1892.
3. December 16, 1892.
4. January 4, 1893.

Unfortunately for the purposes of a strict inquiry into the reasons of our success, two therapeutical agents were simultaneously employed; one was the administration of arsenic in doses of the liquor, increasing from 5 to 15 minims (or $\frac{1}{10}$ grain to $\frac{3}{10}$ grain of arsenious acid); the other was the daily inhalation of oxygen gas, to an amount which corresponded as closely as possible to thirty litres, or a little more than one cubic foot.

I will not here discuss the influence of arsenic in the treatment of leuchæmia. Arsenic is the drug in which hitherto most confidence has been placed, and from which relief is stated to have been obtained; but it is allowed that it must be given in large doses, and that permanent benefit is not likely to result, except in early stages of the disease. That there may be considerable improvement is shown by a case read before the Clinical Society,* in which 90 and 100

* *Clinical Society's Transactions*, 1892, vol. xxv, p. 258.

minims of Liq. Arsenicalis were given daily, but the sequel of the case has yet to be published. In the present instance the arsenical treatment was not meant to be pushed to an extreme, and the daily dose was never more than 45 minims, which was reached twenty-three days after admission; and thirteen days after this it was discontinued, as some toxic effects were becoming manifest.

The oxygen inhalations, to which we may now turn, were continued throughout.

Cases of leuchæmia treated by this method have already been placed on record, and the results have been various. So long ago as 1883 Kirnberger* reported a case in which the inhalation of oxygen was used, on the ground of its value in the treatment of anæmic patients. The patient was a boy aged 10½, who in the late summer of 1882 suffered from loss of appetite, nausea, and pains in the gastric region, and after some preliminary treatment by his mother was seen by a medical man in November. He was then pale, with nausea and tightness of the chest, and presenting hæmic murmurs in the neck and at the heart, but no albuminuria. He became weaker and more pallid, and on December 1 enlarged spleen was found measuring 18 centimetres in length; while the blood presented the leuchæmic condition, with white corpuscles in proportion to the red as 1 : 90. Iron and arsenic were given, but without result. Then oxygen was inhaled to the extent of 30 litres daily, and he immediately began to improve. In ten days he was able to leave his bed for a time; the spleen diminished in size, and, in January, 1883, it was only 14 cm. in length. The blood now showed only an inconsiderable increase of the leucocytes; the red corpuscles were 2,000,000 in the c.mm., and there were numerous microcytes, *débris*, and a great number of abnormally shaped blood-corpuscles, of wedge and tailed form. By the end of January these were less, and the red corpuscles had increased to 3,000,000 per c.mm.; the leucocytes were no longer in excess, and the spleen was only slightly increased. Four weeks later the boy was able to go to school, looking well, but still with a slight enlargement of the spleen. He had taken the arsenic and inhaled oxygen throughout. He was kept under observation, and the red corpuscles never exceeded 4,000,000 per c.mm. In the middle of April there was a fresh enlargement of the spleen, which measured, at the end of the month, 12 cm. in length, and projected two fingers'

* *Deutsch. med. Wochenschrift*, 1883, No. 41, Oct. 10, p. 593.

breadths beyond the ribs. The red corpuscles had fallen to 3,000,000; the wedge-shaped corpuscles were again present, and the hæmoglobin was 30 per cent. of the normal, but there was no increase of the leucocytes. Arsenic without the inhalation of oxygen brought the spleen down to 7 cm. by the beginning of May, but the blood was unchanged, and the boy was still pale. Inhalations of oxygen again restored him to health, and in the middle of September the swelling of the spleen had entirely disappeared, and the blood corpuscles were normal in number. Kirnberger, in his remarks on the case, raises the question of the diagnosis. The proportion of the leucocytes to the red corpuscles was never observed higher than as 1 : 90; and on the relapse of the illness they were not appreciably increased above the normal. It certainly differs in this respect from the typical splenic leuchæmia, of which my own case was an example, and possibly may have belonged to the group of splenic anæmia. Kirnberger himself says: "A doubt might be allowed whether it was to be considered rather as a leuchæmia or pseudo-leuchæmia, since there are authors who only speak of leuchæmia when the increase of white corpuscles is considerable (in the proportion of 1 : 20 or higher); still the name has no importance here, and is a matter of indifference in respect to treatment."

Sticker, three years later,* reports a case treated with oxygen inhalations by Riegel. The patient was twenty-five years of age, and had been ill three years. He had a large splenic tumour, great enlargement of the liver, pain at the lower sternum, œdema, and the well-known appearances of the blood. The proportion of red to white corpuscles was as 1 : 2. On May 5 he began the inhalation of 30 litres of oxygen daily in two portions, and even at the end of a week the red corpuscles had increased and the leucocytes had diminished. On June 19, 60 litres were inhaled, and on August 22 the proportion of the blood corpuscles to one another was nearly normal. The patient's weight had increased from 113 to 126 lbs., and he felt and looked much better. The œdema was gone; the sternum was not now tender; the liver was considerably reduced in size, but the spleen was about two fingers' breadths larger. On September 6 the patient went home, and discontinued the treatment for a time. He returned to hospital and went rapidly downhill, in spite of fresh oxygen

* *Münchener med. Wochenschrift*, xxxiii, 43, 44, 1886. Abst. in *Schmidt's Jahrbücher*, 213, p. 147.

inhalations, and died on January 8, 1886. The autopsy fully confirmed the diagnosis.

Pletzer* mentions two cases treated similarly. In the first the treatment seemed to be favourable, but the patient gave it up after fourteen days. The other patient, who had at the same time disease of the lungs, remained two months under treatment, and was then, at her own wish, discharged in a much improved condition. She felt better; her bodily weight had increased; the liver and spleen were smaller; the proportion of the leucocytes to the red corpuscles was increased from 1 : 5 to 1 : 13. The patient had inhaled 30 litres daily. From examination of the blood this appeared to be a case of purely splenic leuchæmia, whereas in the first patient the implication of the medulla of the bones was both accepted during life, and proved by post-mortem examination, the patient dying eventually from pleurisy.

It cannot be said that either of these cases is completely satisfactory as to the curability of splenic leuchæmia, or as to the curative influence of oxygen inhalations. That the disease in my case did undergo a very remarkable amelioration in the course of nine weeks under the combined influence of oxygen inhalations and arsenical medicines, there cannot be the slightest doubt; and for myself I do not remember to have ever before seen such an improvement in a typical case of splenic leuchæmia. We must not forget, however, that he has since relapsed. At the date of the last information we have of him (August, 1894) the total duration of his illness amounts to over two and a half years, or more than the period—two years—which is accepted as the average duration. But we cannot, of course, say by how much longer he will exceed the average. Many more cases will have to be observed before one can speak with any confidence of the value of this line of treatment. In the meanwhile one may, perhaps, be allowed to make a few remarks, or rather raise some questions as to the theory of its action. We may ask here, as we should ask of the treatment of every disease, whether it really deals with the *materies morbi*, or the *causa causans;* or if it only relieves a symptom, or supplies some deficiency which the disease creates, and, if such deficiency, whether the whole or only part of it.

Kirnberger appears to regard it as supplying the oxygen which the red corpuscles with their deficient numbers are

* *Berliner klin. Wochschr.*, xxiv, 38, 1887. Abst. in *Schmidt's Jahrbücher*, 216, p. 158.

unable to carry to the tissues. If this is the case, it may delay the progress of the disease; it may hinder and delay the results of the disease upon the body generally; and it is conceivable that it may delay them so long as to allow the primary deviation from health to subside; but unless there is some such tendency for the disease to recover of itself in time, the oxygen on Kirnberger's supposition would not have any finally curative effect. In order to discuss the influence of this or any other treatment upon splenic leuchæmia from this point of view, we must know a good deal more of the nature and origin of the disease than appears to be known at present.

Another point of interest is the amount of the remedy that has been employed, and that has been apparently so efficacious, at least temporarily, in some cases. Thirty litres may seem a large quantity, but it is a very small proportion of the amount of air we breathe in a day. Taking the amounts given in Foster's *Text-book of Physiology* for the tidal air (500 c.c.) and the frequency of respiration (17 per minute), we find that 8500 c.c., or $8\frac{1}{2}$ litres are breathed in a single minute, and, therefore, that 30 litres of air can be got through in less than four minutes. The amount of tidal air dealt with in twenty-four hours is 12,240 litres; and if one-fifth of this is oxygen, the amount of oxygen breathed in the day is 2448 litres. The respiration of 30 litres of pure oxygen means the addition of the 24 litres of oxygen, which replace the proportion of nitrogen (four-fifths) in the respired gas. The 24 litres thus added amount to less than one-hundredth part ($\frac{24}{2448} = \frac{1}{102}$) of the amount usually breathed in a day.

It should, moreover, be stated that there was no adequate provision in the apparatus we employed for the complete respiration, without escape, of the 30 litres employed; so that the additional amount of oxygen may be put at a good deal less even than $\frac{1}{100}$. When we consider the great variations in the number of respirations, and the amounts breathed by different individuals, and by the same individuals at different times, it is not at once clear how such results should arise from these means.

As far as I know, there is no particular reason for limiting the amount of oxygen inhaled to 30 litres per diem; and in another case I should certainly give double the amount, as I have recently in a case of splenic anæmia.

Since writing this paper, I have met with a pamphlet by H. Vehsemeyer, of Berlin, who has collected the records of

numerous cases of leuchæmia, and has grouped them according to the method of treatment employed.* He deals first with oxygen inhalations, and shows that more or less improvement, or relief to symptoms, was temporarily obtained in cases published by Sticker, Pletzer, Da Costa, Hershey, Eickenbusch, Sizer, Burschinski, Seguin, and Rehn. On the other hand, it appears that others, as, *e.g.*, Schulze, Stintzing, Mosler, and Curschmann, have seen the method tried in many leuchæmias with no result. He regards Kirnberger's case as one of anæmia and not of true leucocythæmia.

His final verdict is as follows:—"Oxygen inhalations are only to be used with success in the early stage of leuchæmia, as palliative means for the improvement of the general condition and of the condition of the blood; they exercise no influence upon the enlargement of the spleen, and cannot favourably affect either the progress of morbid process or the second stage of the disease." From my own case I am disposed to take a more favorable view of their influence upon the spleen.

* *Die Behandlung der Leukæmie: Kritische Studie*, von Dr. H. Vehsemeyer, Berlin, 1894.

XI.—*A case of Supernumerary Testis.* By W. Arbuthnot Lane, M.S. *Read November* 23, 1894.

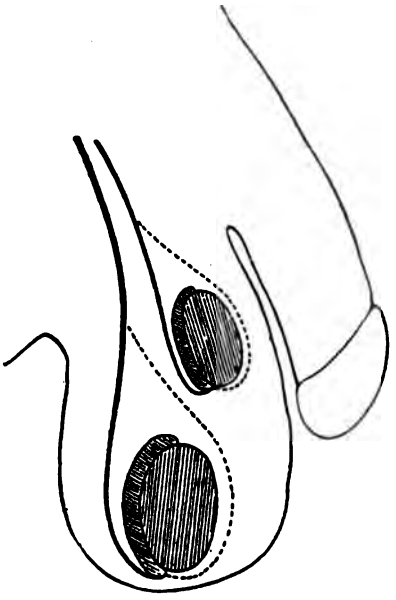
C. G., aged 17, a black-haired, dark-skinned lad, well developed for his age, was admitted into Guy's Hospital under my care on August 15, 1894. He complained of a rounded lump in the right half of the scrotum, which was discovered in July by a medical board who examined him as a candidate for the naval service, and who refused him on that account. He was unable to say how long the swelling had existed, as he had never noticed it before. He desired to be relieved of it.

The lump he complained of was as large as a good-sized cherry, with a smooth surface, having apparently some connection with the cord by its posterior aspect. It had no connection with the right testicle, and could, by manipulation, be separated from it by an interval of about two and a half inches. Traction in a downward direction on the testis, so as to render the spermatic cord as tense as possible, did not affect the range of mobility of the mass. Moderately firm pressure produced no pain, and severe pressure caused but slight discomfort. On being interrogated after the operation whether the feeling experienced from this pressure resembled that produced by compressing the normal testis, he said there was no similarity. This portion of his evidence was obviously not very reliable. The left testis was well developed and of full size, while the right one was considerably smaller. If the bulk of the right testis were increased by that of the lump, the whole would still be smaller than the left one. Both right and left testes were apparently perfectly normal in their structure.

The tumour was exposed by an incision into the right half of the scrotum. It was found to be attached to the testis by a fascial mesentery, which was about three inches in length, in whose upper free margin a small rounded cord could be felt blending above with the spermatic cord. This proved, on subsequent examination, to be the vas and vessels of the supernumerary testis. The tumour was freely incised, and a capsule was turned back with some difficulty from its surface. It was then discovered that this capsule was the visceral

layer of the tunica vaginalis with the tunica albuginea, and that the organ resembled exactly a small testis in structure, in that it possessed a body, tunica vaginalis, epididymis, and vas. The other mass, which had been regarded as the normal testis on the same side, was accordingly turned out through the incision, and its structure carefully examined. It proved to possess all the components of a normal testis. It was replaced in the scrotum, and the supernumerary organ, which was too much mutilated to be of any use, was removed.

FIG. 2.



Represents diagrammatically the apparent size and relations of the two testes to one another, the dotted lines indicating the parietal layer of the tunica vaginalis of each.

The following is the report of the Surgical Registrar, Mr. Bellingham Smith, on the tumour:—"When seen the organ had been placed in strong spirit, and had had both layers of the tunica vaginalis peeled off it. The body of the testis was oval, slightly lobulated, about $\frac{7}{8}$ inch long by $\frac{3}{4}$-inch broad. It possessed a tunica vaginalis. The epididymis was situated at the back part of the testis, and was about $\frac{3}{16}$ inch thick. The vas was cut through close to the tail of the epididymis. The sections made through the body showed a structure radiating from the mediastinum. The spermatic tubules were readily teased out. Microscopical sections showed well-formed tubules with spermatogenesis proceeding."

This condition of supernumerary testis is, as far as I know, one of extreme rarity. The relatively smaller size of the right testis supports the view that the third organ is developmentally a subdivision of it in the same manner that is observed occasionally in the case of the kidney.

XII.—*A series of eighty cases treated with Diphtheria Antitoxin, with observations on their Bacteriology.*

By J. W. Washbourn, M.D., E. W. Goodall, M.D., and A. H. Card. *Read December* 14, 1894.

On October 22, through the kindness of the Council of the British Institute of Preventive Medicine, the Eastern Hospital was provided with a supply of antitoxin prepared by Dr. Ruffer, and a promise was given that the supply would be continued until we were enabled to form an opinion of the efficacy of the remedy.

The treatment was commenced on the following day, and was carried out continuously until November 27, by which time we considered that sufficient cases had been treated to form an opinion. As we believe that a sufficient interval has elapsed since the treatment was commenced in the last patient of the series, and as we are aware of the interest taken in the subject, we have thought it well to give to the members of the Clinical Society an account of our experience.

Bacteriological examination.—A bacteriological examination was made of all the cases directly they were admitted into the hospital. The *method* employed was as follows. The tongue was depressed with a spatula, and a little of the exudation was removed from the throat by means of a previously sterilised platinum rod. When there was no exudation the rod was simply rubbed over the surface of the fauces, and in cases of laryngeal diphtheria without angina was passed down the throat as near to the larynx as possible. Tubes of coagulated blood-serum were inoculated with the platinum rod, and the inoculation was always made by the same observer (A. H. C.), so as to obtain as far as possible similar results. In making the inoculation the surface of the tube was first moistened with the water of condensation, the tube was then inoculated and the surface again moistened. In this way the growth was evenly spread over the surface, and the colonies were separate. At first three tubes were inoculated from each case, but we afterwards found that two tubes were quite sufficient. At the same time as the tubes were inocu-

lated, cover-glass preparations were made from the exudation, and were examined microscopically. In about a third of the cases the diagnosis could be made from this alone; in one case the diphtheria bacilli were present in enormous numbers in the exudation, and almost in a state of pure cultivation. The tubes were placed in an incubator at 37° C., and were examined in about twenty hours, and cover-glass preparations from the different colonies were made. Sometimes the examination was made a few hours earlier, sometimes a few hours later. After the first examination one of the tubes was again placed in the incubator, and was examined from day to day to see if fresh colonies developed.

Attention was paid to the following points:—The number of typical and atypical diphtheria colonies; the variety of the diphtheria bacillus; and the number and character of the other colonies present. Special attention was paid to the latter point on account of the importance that has been attached to the association of other bacteria with the diphtheria bacillus upon the clinical course of the disease.

Diagnostic value of the bacteriological examination.—In the majority of cases of true diphtheria the naked-eye appearances of the cultivations were characteristic, the diphtheria colonies, at the end of twenty hours, being opaque, and of the size of a pin's head. The only colonies resembling them were those of a coccus described by Roux and Yersin, and called by them the Brisou coccus. They differed from the diphtheria colonies in being more transparent and less prominent; all doubt was removed by a microscopical examination. In addition to the typical colonies other diphtheria colonies, much smaller and more transparent, frequently developed.

In eight out of the sixty-one cases of true diphtheria none of the colonies were typical; they either did not assume the characteristic appearances at the usual time, or were smaller than usual, or differed in some other respect. Such colonies we have called "atypical." We have experimental evidence to show that they were true diphtheria bacilli.

Of the eighty cases which were examined, and which were all certified to be suffering from diphtheria, diphtheria bacilli were found in sixty-one. The percentage of cases that were not true diphtheria was thus 23·75. It is an interesting coincidence that in the memoir of MM. Roux and Yersin in the *Annals of the Pasteur Institute* exactly the same number of cases was examined with exactly the same result.

In one of our cases of croup there was no exudation

upon the fauces, yet a bacteriological examination revealed the presence of diphtheria bacilli. The case ultimately required tracheotomy, and recovered.

With one exception all the cases in which diphtheria bacilli were found were also diagnosed from the clinical point of view as diphtheria. The exception was a case which had the clinical aspect of follicular tonsillitis.

On the other hand, there were eleven cases which presented, on admission, the clinical aspect of true diphtheria, but in which no diphtheria bacilli were present. In most of these cases the amount of exudation was slight, but in one there was abundant exudation. A second examination of this case still gave negative results as far as diphtheria bacilli were concerned.

The further clinical course of these cases confirmed us in the opinion that they were not true diphtheria. The only one amongst these patients to die was suffering from bronchitis, and to this cause death was attributed.

The result of our observations in this series of cases fully coincides with our previous experience of the extreme importance of a bacteriological examination in all cases of angina and croup. It not only enables non-diphtherial cases to be eliminated, but it also reveals the true nature of cases which would otherwise not have been considered to be diphtheria.

Prognostic value of bacteriological examination in true diphtheria.—We have endeavoured to ascertain whether any prognostic value could be attached to the results of the bacteriological examination of the cases of true diphtheria. The cases from a clinical point of view were independently divided into three categories,—mild, moderately severe, and severe. The relative proportion of these was calculated, and we could thus be certain whether any series showed a marked deviation from the average.

Microscopical examination of the exudation.—Cover-glass preparations of the exudation were carefully examined, and the following points were noted :

1. The number of bacteria present, whether few or many.
2. The character of the bacteria present, and a rough estimation of their several numerical strengths.

In the majority of cases a large number of bacteria were found. In six cases the exudation was remarkably free from bacteria, and of these cases four were mild and two of moderate severity. In ten cases the exudation was com-

paratively free; half of these cases were mild, and only one severe; none of them were fatal.

From these figures we conclude that the fewer bacteria in the exudation, the less severe is the case likely to be.

As to the character of the bacteria found in the exudation, they were of various kinds, and included straight bacilli of various sizes, comma-shaped bacilli, fine spirilla, diplococci, streptococci, and other micrococci.

Diplococci were almost always abundant, and were often grouped in large masses, especially on the surface of epithelial cells. In only seven cases were they absent.

In twelve cases fine spirilla were present, sometimes in large numbers.

In twenty-two cases comma-shaped bacilli were present, and at times in abundance. In about half the cases straight bacilli of various sizes were present.

No prognostic value could be attached to the absence or presence, or to the number, of the diplococci, spirilla, straight or comma-shaped bacilli.

Particular attention was paid to the streptococci. In more than half the cases some were found in the exudation. In seven cases many were found; and of these four were mild cases, two severe, and one moderately severe. Careful analysis of these, as well as of the cases in which a smaller number of streptococci were found, showed that they were neither more nor less severe than the average of the cases.

In about a third of the cases characteristic diphtheria bacilli could be distinguished in the exudation with sufficient certainty to make a diagnosis of the nature of the disease. Very few of these cases were of a mild type, and more than a third of them died; so that the finding of characteristic bacilli in the exudation in any quantity is of unfavorable import.

Examination of the cultivations.—We will now turn to the examination of the cultivations, and see what information can thus be obtained.

Cases in which only diphtheria colonies developed.—In thirteen cases no other colonies than those of diphtheria developed in the tubes even after three days' incubation. The majority of these cases were of a severe type, and a third of them died.

Cases where only a small number of diphtheria colonies developed.—In seventeen cases only a comparatively small

number of diphtheria colonies developed in the tubes. Eleven of these cases were mild and only four were severe.

Cases where the diphtheria colonies were atypical.—In eight cases the diphtheria colonies were atypical. Of these cases five were mild, two severe, and one moderately severe.

It would thus appear that when only diphtheria colonies develop in the tubes the case is likely to be severe; but that when the diphtheria colonies are few in number, or atypical in character, the case will probably be mild.

Cases where the diphtheria colonies were associated with streptococci.—In forty-five cases colonies of streptococci developed in addition to those of diphtheria; but in only eighteen were they present in large numbers. Of the eighteen cases ten were mild, and four were severe; only three died.

In the series of sixty-one cases of true diphtheria there were three cases which were remarkable for containing a very large number of streptococci, both in the cover-glass preparations of the exudation and in the cultivations. Two of these cases were mild; the other was severe and ended fatally, but was complicated with scarlet fever. In one of the mild cases many diphtheria colonies of the long variety developed, and in the other a moderate number also of the long variety.

The question of the association with the streptococcus is one of great importance; and it is stated by several foreign observers, especially by Roux, Martin, and Chaillou, that such an association is most unfavorable. Our observations certainly show that an association with streptococci as evidenced by a single bacteriological examination is not of unfavorable import; in fact, our cases lead to the opposite conclusion.

We are unable to give a satisfactory explanation of this want of agreement with other observers. It was not caused by any difference in the variety of the diphtheria bacillus; for in seventeen of the eighteen cases the bacillus was of the long variety, and in one of the medium variety. In the eighteen cases the colonies of streptococci many times outnumbered those of diphtheria.

Cases where the diphtheria colonies were associated with those of the Brisou coccus.—Three cases were associated with the Brisou coccus; two were mild, and the other moderately severe.

Cases where the diphtheria colonies were associated with the

Staphylococcus aureus.—Two cases were associated with the Staphylococcus aureus; both were mild.

The variety of the diphtheria bacillus.—In three cases only the short variety of the diphtheria bacillus was found; all were mild.

In ten cases the medium variety was found; half of the cases were mild. In the rest of the cases the bacilli were of the ordinary long variety. It would thus appear that the short variety is the least virulent, and that the medium variety is less virulent than the long.

Bacteriological examination of non-diphtherial cases.—In nineteen cases no diphtheria bacilli were found. The microscopical characters of the exudation were similar to those in the diphtherial cases, with the exception that diphtheria bacilli were absent. In one case colonies of streptococci alone developed in the tubes; in another case only colonies of the Brisou coccus. The remaining cases showed a mixture of these two micro-organisms in varying proportions. No other kinds of bacteria developed.

In one case which was fatal from bronchitis the exudation contained two kinds of bacteria; one was a large bacillus which stained irregularly, and the other was a short bacillus united in pairs. In the cultivations many Brisou colonies developed, and a few streptococci, but neither of the bacilli grew.

Clinical Observations.

Mode of injection.—The serum was injected in nearly every instance into the subcutaneous tissue of the flank, as, after trial, it was found that it was much more easily injected into this place than into the buttock. The syringes employed were similar to the one described and figured in the *Lancet* for December 8, p. 1355; one of them held about 25 c.c., the other 10 c.c. The washer of the plunger is made of asbestos, and can be tightened up when in place by means of a screw at the end of the piston rod. It is best not to fix the needle directly on to the nozzle of the barrel, but to connect it by means of a piece of india-rubber tubing, so as to prevent injury in case the patient moves. Immediately before giving an injection the skin was washed, first with soap and water and then with a solution of carbolic acid; and the syringe, needle, and tubing were boiled. If these simple antiseptic precautions are taken, and the serum be pure, there is no risk of inflammation; and that these precautions were thoroughly

carried out is shown by the fact that in not a single instance out of 182 separate injections, was there at any time the slightest sign of any local inflammation.

The amount of serum injected at one time has varied from 5 to 30 c.c., the latter in two doses of 20 and 10 c.c. The total amount injected during the course of any one case has varied from 5 to 90 c.c., the latter in eight separate injections on successive days. A common dose has been 20 c.c., followed by another 10 c.c. in from eighteen to twenty-four hours. With a well-made syringe as large an amount as 20 c.c. can be injected without any difficulty. A local swelling is formed, which disappears in about twenty minutes. Further remarks on the question of dosage will be made later.

1. *Statistics.*—In all eighty-two cases were treated. They were all under the age of fifteen years, and were sent to the hospital certified to be suffering from diphtheria. Two were found on the day after admission to be cases of scarlet fever, and have been excluded from the following statistics because similar cases have been omitted from the figures obtained for the sake of comparison from past records. We have, therefore, eighty cases which on admission to the hospital were presumably diphtheria. Now of these eighty cases, eight would have been put down, from clinical observation extending over a few days, as cases of angina other than diphtheria. The clinical diagnosis of these eight cases was confirmed by the bacteriological evidence. The remaining seventy-two cases were, with only one exception, clinically diphtheria. It is this series of seventy-two cases which we compare with previous cases diagnosed clinically as diphtheria.

We are obliged to make the clinical and not the bacteriological diagnosis the basis of a comparison for the simple reason that a bacteriological examination has in the past been made in only a very few cases. Our comparison series go back to the beginning of 1893; from that time to the present the diagnosis of the cases has been supervised by one of the writers (E. W. G.), so that there is no question of the introduction of many varying personal equations. Again, it is found that the number of cases recognised clinically to be not diphtheria among the patients under fifteen years of age admitted to the hospital from the beginning of last year to the date of commencement of the antitoxin treatment was 10·4 per cent.; which corresponds with the eight out of the eighty cases with which we are

dealing. Hence we may fairly take the remaining seventy-two cases which were put down from a clinical point of view as being diphtheria, and compare them with similar previous cases.

Of these 72 cases 14 have died, which gives a mortality of 19·4 per cent. The mortality amongst children under fifteen admitted into the Eastern Hospital during 1893 was 41·8 per cent. (397 cases with 166 deaths), and during 1894 up to October 22, 36 per cent. (400 cases with 144 deaths): a total of 797 cases with 310 deaths, with a mortality of 38·8 per cent. Of the 72 cases immediately preceding those treated with antitoxin 28 died—a mortality of 38·8 per cent.

Case mortality of diphtheria in children under fifteen at the Eastern Hospital.

	Cases.	Deaths.	Mortality per cent.
1893	397	166	41·8
1894 (Jan. 1—Oct. 22)	400	144	36
Jan. 1, 1893—Oct. 22, 1894	797	310	38·8
Sept. 14—Oct. 22, 1894 (39 days) ... Not treated with serum.	72	28	38·8
Oct. 23—Nov. 27, 1894 (36 days) ... Cases treated with serum.	72	14	19·4

There is thus a marked difference in the case mortality of the cases treated with antitoxin and those not so treated; in fact, the one is just half the other. It may, of course, be suggested that the seventy-two serum-treated cases were perhaps of an abnormally mild type. But, in the opinion of the one of us who is most familar with the nature of the cases admitted into the hospital, these cases were rather above than below the average severity.

With regard to the comparison of our mortality with that of other hospitals there is the difficulty of the varying standard of diagnosis; so that we cannot lay too much stress upon such a comparison. Through the courtesy of Drs. Mac Combie, Bruce, Caiger, and Matthews, we have been furnished with the following statistics.

During the period from October 23 to November 27 inclusive there were admitted into the Western Hospital 60 cases with 20 deaths, a mortality of 33·3 per cent.; into the South-Western 59 cases with 19 deaths, a mortality of 32·2 per cent.; and into the South-Eastern 38 cases with 9 deaths, a mortality of 23·6 per cent. Now in these hospitals, with the exception of the last, the mortality was over 30 per cent.;

but at this hospital the number of cases is too small for an accurate comparison; and we understand from Dr. MacCombie that during the whole year the cases admitted into the hospital have been mild, and that the mortality at all ages has been only 25 per cent. as compared with a mortality of 39·9 per cent. last year. If we add the cases at these three hospitals together we get a mortality of 30·5 per cent.

At the Fountain Hospital we have only been able to obtain the mortality for the whole of the months of October and November. During this period there were 123 cases with 38 deaths, a mortality of 30·8 per cent.

All these statistics refer to patients under fifteen years of age.

But though these seventy-two cases were *clinically* diphtheria, in eleven of them the diagnosis was not confirmed by a careful bacteriological examination. One of the eleven was a fatal case, and is included among the fourteen deaths mentioned; for it would certainly have been considered as diphtheria had there been no bacteriological examination. The patient was a child eleven months old, who was also the subject of acute bronchitis, which was the immediate cause of death. There was an irregularly distributed thin membranous exudation upon the fauces, much like what is seen in diphtheria. Subtracting these eleven cases with one death, we have sixty-one cases of true diphtheria with thirteen deaths, a mortality of 21·3 per cent. So that even the mortality of the true diphtheria cases only, is considerably lower than that (38·8 per cent.) of the previous series of clinically diphtheria cases.

In ten of the fatal cases the cause of death was the direct action of the diphtheria poison without complications, and one of these cases died within eight, and the other within thirty-two hours of admission. In the eleventh the patient, who was, on admission, also the subject of scarlet fever, died of dyspnœa some days after tracheotomy. At the autopsy there was much pulmonary emphysema. The lower end of the tracheotomy tube had caused ulceration of the wall of the trachea, exposing the cartilage of three of the rings. The tracheotomy wound was fairly healthy. There had been during life some cellulitis of the upper extremities. In the inflammatory exudation streptococci were found in abundance; they had also been found in the exudation on the fauces. The twelfth patient—a severe case of post-scarlatinal diphtheria—was tracheotomised, and was doing well, until the escape of some vomit into his lung set up a septic pneu-

monia, of which he died on the thirtieth day of his illness. The thirteenth case proved fatal from scarlet fever occurring a week after admission. It was also a severe case of diphtheria in which tracheotomy had been performed, and, though progressing favorably, the patient was by no means out of danger when the attack of scarlet fever supervened.

There were amongst our cases fifteen in which the larynx was affected, and in nine of these tracheotomy was necessary and was performed. Three of the patients died, six recovered. In thirteen series of nine cases of tracheotomy extending back to the beginning of 1893 the number of recoveries varied from none to four; the average number of recoveries per series of nine being 1·75. The cause of death in the three cases of tracheotomy has already been mentioned. Of the six laryngeal cases in which no operation was performed two died, both from the toxic effects of diphtheria.

For the sake of comparison with M. Roux's cases we have analysed our cases of true diphtheria, and have divided them into the following categories: Anginas, Croups not operated on, Tracheotomies.

ANGINAS.

Angina pure (*i.e.* without association with other bacteria).—Thirty cases, with eight deaths, a mortality of 26·6 per cent.

Angina associated with streptococci.—Eleven cases, with one death, a mortality of 9 per cent.

Angina associated with the Brisou coccus.—Three cases, no deaths.

Angina associated with streptococci and with the Staphylococcus aureus.—Two cases, no deaths.

CROUPS NOT OPERATED UPON.—Six cases, with two deaths.

Pure diphtheria.—Four, with two deaths.
Associated with streptococci.—One, with no death.
Associated with the Brisou coccus.—One, with no death.

TRACHEOTOMIES.—Nine, with three deaths.

Pure diphtheria.—Five, with one death.
Associated with streptococci.—Three, with two deaths.
Associated with streptococci and Staphylococcus aureus.—One, with no death.

In comparing these with M. Roux's statistics we find remarkable differences.

Of pure diphtherial angina there were in his series 120 cases, with nine deaths, a mortality of 7·5 per cent.

Of angina associated with streptococci thirty-five cases, with twelve deaths, a mortality of 34·2 per cent.

It will thus be seen that our pure angina cases showed a much higher mortality, and those associated with streptococci a much lower mortality than M. Roux's. We have already alluded to similar conclusions in the account of the prognostic value of the bacteriological examination. On the other hand, our cases associated with the Brisou coccus agree with his in being mild.

The number of tracheotomies and croups is too small for conclusions to be drawn.

2. *Clinical evidence.*—The statistics we have brought forward offer a strong argument in favour of the beneficial results of the treatment by antitoxin, but they are not the only argument. There is the clinical evidence.

Of the sixty-one cases of true diphtheria twenty were severe cases, exhibiting symptoms of poisoning by diphtheria at the time of admission, cases in which an unfavorable prognosis was given. Eight of them recovered.

Of these twenty, three were cases of faucial diphtheria, with two recoveries; twelve were faucial and nasal, with five recoveries; one was faucial and laryngeal, and was fatal; four were faucial, laryngeal, and nasal, with one recovery.

In severe cases the effects of the remedy are not observed until two or three days have passed after the first injection. In moderate and mild cases the effect is usually observed the next day or the next day but one. The membrane or exudation may even spread or become thicker during the first day or two after the commencement of the treatment. The signs that the antitoxin is exercising a good effect are (i) a lessening in the amount of the membrane; (ii) a notable fall in the pulse-rate; (iii) an improvement in the general condition of the patient; and (iv) a fall of temperature in cases where the temperature has been raised.

When once the exudation begins to disappear, it continues to do so in our opinion with greater rapidity than is usual under ordinary methods of treatment. The pulse-rate does not fall with the same rapidity with which the membrane disappears. In some cases a rapid decrease in the size of swollen cervical glands, and disappearance of cervical cellulitis, have been observed.

That this clinical recognition of the effects of the serum

was not mere fancy and imagination on our part, we beg to offer the following piece of evidence. We did not know whether the serum supplied to us up to November 13 came from the same or from different horses. On November 13 a fresh supply was received, and in a few days it plainly appeared to the one of us who was most immediately concerned in the treatment of the patients that this fresh serum was considerably more efficacious in its antitoxic action than that which had previously been used. Accordingly the dose of the remedy was diminished, and a letter was written to Dr. Ruffer, telling him that this had been done, and the reason. A letter was subsequently received from him to say that the clinical quite bore out the experimental evidence, for that the serum supplied to us on November 13 came from a different horse from that of the former supply, and was at least five times as strong, bulk for bulk. We have since been informed that the first lot of serum came entirely from horse No. 1, and the supply after November 13 from horse No. 2.

The next point we consider is the effect of the serum on complications. In our opinion it has no effect on the prevention of such complications as are not due to the direct action of the poison of diphtheria, *e. g.* secondary adenitis, lobular pneumonia, &c.; except perhaps in this way, that the more quickly a patient gets well, the less likely is he to have complications. We have noticed no diminution in the incidence of albuminuria; thirty out of the seventy-two patients had albuminuria on two or more occasions; that is, 41·6 per cent. Every one of these thirty instances occurred in cases of true diphtheria; there was not a single instance amongst the non-diphtherial patients. It should be added that, when possible, the urine was tested every day. With respect to paralysis it is still premature to speak, for it is at the time of writing only sixteen days since the last patient of the series was admitted to the hospital. But so far six cases have occurred, and none of them have been severe. Six out of seventy-two is an incidence of 8·3 per cent. But this incidence will probably be higher. The incidence of paralysis amongst the 797 patients previously referred to was 13·7 per cent.

The next question is, Has the serum any effect of its own apart from its antitoxic action? We have observed two effects. The first is a rash; this has followed in twenty out of the eighty cases—25 per cent. The rash has appeared at from the seventh to the nineteenth day after the first injec-

tion. Its form is variable. In some cases it has consisted of numerous patches of gyrate erythema, in others of patches of urticaria, in others again of ill-defined blotches, and in one or two of a diffused erythema something like scarlet fever. When on the face it causes a little puffiness. It may last a few hours or a few days. It may be limited to the extremities or involve the skin of the whole of the body. It usually appears first about the wrists and ankles. Sometimes the temperature is raised, at others not; but whether there be pyrexia or not, or whether there be much or little rash, the only inconvenience the patients have experienced has been itching. A well-marked rash with pyrexia has followed a single dose of 10 c.c. We have not seen it follow a smaller dose. The other effect has been pains in certain of the joints. We have observed six such cases. The smallest dose giving rise to this condition was 10 c.c. The pains came on from eight to nineteen days after the first injection. The joints affected have been chiefly the hips, wrists, and ankles. In all six cases the hips have been affected, in three the wrists, in two the ankles, in one the elbows and one sterno-clavicular joint, and in one some of the fingers. In one case there was slight and transient swelling of one of the wrist-joints, in another of some of the finger-joints. Otherwise pain was the only local symptom. The pain was aggravated by movement. It was first noticed in five of the cases in the hips, in one in the wrists. The pain lasted from one to three days. There was at the same time pyrexia, and except in one case, one of the varieties of the rash already described. There was no cardiac affection. Salicylate of soda gave no relief whatever, but the administration of two or three small doses of Tinctura Opii, and wrapping the joints in warm cotton wool gave alleviation. In none of the cases did the condition of the patient become serious from this complication. Two or three cases of joint pains have been recorded in a recent number of the *Deutsche medicinische Wochenschrift.*

In connection with these rashes and joint pains we have made the following observation. We have mentioned that we were supplied with serum from two horses; with the serum from horse No. 1, forty-five patients were treated; with serum from horse No. 2, thirty-five. Sixteen cases of rash and five of joint pains have occurred amongst the forty-five patients treated with serum No. 1; while up to the present only four cases of rash and one of joint pains (the slightest that has yet occurred, only the left hip being affected) have arisen amongst the thirty-

five patients treated with serum No. 2. As we have already mentioned, serum No. 2 was very much more potent in its antitoxic action than serum No. 1, and it was not necessary in most cases to inject so large a quantity. So that it is possible that these effects may have been due to the quantity of serum injected.

With regard to treatment, two or three of the patients had a little brandy given them on their admission to the hospital on account of prostration, and complications were treated *secundum artem*. But the only local treatment that has been employed has been the occasional flushing out of the mouth, fauces, and nasal passages with warm water in the very severe foul cases.

We have, from the evidence we have brought before you, come to the conclusion that antitoxic serum is a remedy of great value in diphtheria; and to this conclusion we have come as much from clinical observation as from statistical evidence.

With respect to dosage, we will only speak of serum No. 2. This serum is of such a strength that ·0001 c.c. of it protects guinea-pigs injected with a dose of diphtheria toxin, which is fatal in thirty-six hours or less to a control animal. With serum of such a strength we should recommend, for a severe case, 20 c.c. when the patient is first seen, followed by 10 c.c. in from eighteen to twenty-four hours; and again another 5 or 10 c.c. in another eighteen to twenty-four hours; for a moderately severe case a first dose of 10 c.c. followed by one of 5 c.c. the next day, and perhaps another 5 c.c. the day after that; for a mild or doubtful case one dose of 5 c.c. should there be any reason to suspect that the case is likely to become worse.

Because in any given case the larynx is involved we do not necessarily consider the case a severe one; for by severe we mean one in which the toxic symptoms of diphtheria are present. But seeing with what rapidity membrane will spread along the air-passages, we recommend that from 5 to 10 c.c. of serum should be given in every case of diphtherial croup without toxic symptoms, to be followed by another 5 c.c. the next day if the symptoms show no improvement. As far as our cases go we find that if in a severe case there is no improvement by the fourth day of the treatment, none is likely to ensue. But where improvement is observed by that time, we think it is well to give another injection of 5 c.c. on the fifth and sixth days.

We recommend the amount of the dose to be regulated not by the age or weight of the patient, but by the severity of the attack.

The Cases which were not Diphtheria.

The nineteen cases of disease which were shown by bacteriological examination not to be diphtheria were as follows:

Simple inflammation of the fauces, six, of which one was complicated by stomatitis, and another by cervical adenitis and a discharge from the nose.

Membranous inflammation of the fauces and bronchitis, one.

Follicular tonsillitis, two.

Acute tonsillitis, six, one with discharge from the nose.

Simple ulceration of the tonsil, two, in one of which there was a nasal discharge.

Simple ulceration of the soft palate, one.

Croup, one.

The case of membranous inflammation of the fauces and bronchitis was in a most grave condition when admitted, and proved fatal the next day. All the other cases recovered, and were in the hospital from one to five weeks. None of them contracted diphtheria, even though they remained in wards allotted to diphtheria, in which there were ten or eleven patients affected with that disease. This immunity, however, we in no way attribute to the antitoxin which they received. In the course of the year many cases of sore throat other than diphtheria are admitted into the diphtheria wards, but it is rare for any of these cases to develop diphtheria. If in a diphtheria ward the beds are not placed too close to one another, the ventilation is good, the drinking-vessels, spoons, spatulæ, &c., are properly cleaned and disinfected, and the patients, especially those who are allowed up, are sufficiently supervised, there is practically no risk of a case of simple sore throat, whatever its nature, becoming converted into one of diphtheria.

In conclusion we beg to offer our best thanks to the Council of the British Institute of Preventive Medicine for so generously allowing us to be supplied with the serum; to Sir Joseph Lister, the President, for his kind advice and encouragement; and to Dr. Ruffer, the Director, to whom is due the introduction into this country of the antitoxic treatment of diphtheria. We are also indebted to Dr. Richards,

Assistant Medical Officer at the Eastern Hospital, for help in carrying out the treatment and in recording the notes of the cases; and to the Secretaries of the Clinical Society for affording us the opportunity of reading our paper this evening.

Note added March 19, 1895.—One of the seventy-two patients, a boy aged two years, subsequently died, thirty-seven days after admission, of pyo-pneumothorax, secondary to tuberculous disease of the lungs, symptoms of which had been present for some time before diphtheria supervened. There were no more cases of joint-pains, and only one of rash, which occurred in a patient treated with serum from horse No. 2. No further instances of albuminuria occurred. There were three more cases of paralysis, making a total of nine, an incidence of 12·5 per cent.

The following table shows the ages of the seventy-two patients:

Under 1 year,			4,	of whom	1	died.
1	to	2 years,	10,	„	2	„
2	„	3 „	7,	„	1	„
3	„	4 „	9,	„	3	„
4	„	5 „	10,	„	5	„
5	„	10 „	22,	„	2	„
10	„	15 „	10,	„	0	„

Further information on the question of age may be found in the *British Medical Journal* for January 12, 1895, p. 100.

With respect to the question of the time at which the treatment was commenced, of thirty-two cases in which it was begun on the first, second, or third day of the disease, two died; while of twenty-nine, in which it was begun on the fourth or later days, eleven died. All these sixty-one cases were demonstrated bacteriologically to be diphtheria.

XIII.—*Some cases of Rapid Cure of Diphtheria by means of antitoxin.* By W. Henry Kesteven. *Read December* 14, 1894.

L. K., æt. 12. This lad is not very strong; his health has not been satisfactory for some months past.

On Saturday, November 10, he seemed very seedy, and his temperature was found to be 101°, pulse 120. No signs of throat mischief.

Sunday, November 11.—Left tonsil swollen. Gave him 5-grain doses of sodium salicylate every four hours. The temperature was 100°, pulse 110.

Monday, November 12.—Temp. 99·6°, pulse 100. White patches appearing on tonsil.

Tuesday, November 13.—Patches on tonsil coalesced at superior portion of tonsil. Recognised it as a continuous diphtheritic membrane. Opinion confirmed by Mr. Butlin. Temp. 101·2°, pulse 120.

Wednesday, November 14.—Temp. 100°, pulse 96 at 6 P.M. Injected antitoxin, 12 c.c., at 6.30. Temperature and pulse not altered.

Thursday, November 15.—9.45 A.M., temp. 100°, pulse 104. 2.15 P.M., temp. 100°. 6.40, temp. 98·8°, pulse 88. 6.50, injection antitoxin, 10 c.c. 7.15, temp. 98·6°, pulse 86.

Friday, November 16.—10 A.M., temp. 100°, pulse 84. 6 P.M., temp. 100°. Membrane disappearing.

Saturday, November 17.—8.30 A.M., temp. 99·4°. 5 P.M., temp. 100°.

Sunday, November 18.—11.45 A.M., temperature normal. All the membrane gone. 5.30 P.M., temperature normal.

Throughout the boy has taken food well. There has been no albumen in the urine. At the time of the first injection, which, in consequence of an accident, was only 12 c.c., the membrane entirely covered the left tonsil. The right tonsil was swollen and red, and there were traces of membrane appearing thereon. The injection caused a swelling on the abdomen about the size of a small hen's egg. It disappeared within half an hour. Next morning there was a little hardening and redness where the needle had been inserted, but this also had gone before the second injection was given. There was no rash.

W. K., æt. 11.

Monday, November 12.—The boy seemed ailing, but not bad enough to stay from school.

Tuesday, November 13.—Distinctly unwell, tonsil swollen and red; glands in neck painful. Temp. 101·2°, pulse 100. Five grains of sodium salicylate every four hours. Seen by Mr. Butlin in evening, at which time membrane had begun to form.

Wednesday, November 14.—Temp. 100·1°, pulse 96. Membrane spreading over front of the left tonsil. Bowels relaxed. Temperature before injection as above. 7.30, injection of antitoxin, 15 c.c. No change in temperature.

Thursday, November 15.—9.45 A.M., temp. 101·2°, pulse 116. 2.15 P.M., temp. 101·2°. 6.15 P.M., temp. 101·6°, pulse 120. 6.30 P.M., injection, 10 c.c. antitoxin. 7 P.M., temperature and pulse the same.

Friday, November 16.—10 A.M., temp. 101·2°, pulse 96. 6 P.M., temp. 100·8°. Membrane visibly diminished.

Saturday, November 17.—8.30 A.M., temperature normal. 5 P.M., temperature normal. Slight erythematous rash on chest.

Sunday, November 18.—9.15, temperature normal. Membrane disappeared. 5.30, temperature normal.

Monday, November 19.—Rash scarcely visible.

There has been no albumen all through.

H. H., æt. 8½.

November 9.—Taken ill on November 9th, 1894. By the evening of the 9th the tonsils were very much enlarged, with a grey patch on the right one; tenderness and swelling of the glands of the neck.

November 11.—The membrane is rapidly spreading, almost all the uvula is covered and both tonsils. Temp. 102·4°.

November 12.—Patient had a very restless night, membrane extending round the soft palate. Great difficulty in swallowing. Temp. 101·8°.

November 13.—Patient very ill. Has a quantity of mucus from the nose, causing discomfort, almost closing the nostrils. Membrane still spreading. Voice a little husky. Temp. 99°.

November 14.—Patient much worse. Has great difficulty of breathing, and complains of pain in the left ear and throat, he is also very restless, and has still a great quantity of mucus from the nose. Temp. 100°.

November 15.—Patient still remains in the same condition. Profuse discharge from the nose and throat. Left ear pain-

ful. 10 P.M., 15 c.c. antitoxin serum injected into the left buttock, causing a slight swelling for a short time. Beyond the prick of the needle, patient felt very little discomfort. Temp. 100° at time of injection (10 P.M.).

November 16.—Patient slept eight hours during the night. At 2 A.M. temp. 99·2°. Complains of tenderness at the site of the injection. Breath very offensive. Temp. 99·4°.

November 17.—Patient very low, but has less mucus coming from the nose; glands not so tender, and less swollen. Breathing rather bad, and cough troublesome. Temp. 98·8°.

November 18.—Patient had a restless night, but on the whole is very much better. A large piece of membrane has been discharged from the nose. Breathing much better, membrane also clearing off the throat, and less mucus from the nose. Patient in slight perspiration. Temp. 98·8°.

November 19.—Patient slept ten hours during the night, and is much better. Throat cleared of membrane. Farinaceous diet. Subnormal temperature.

November 20.—Patient feels quite well and is perspiring freely. Still has a little mucus from the nose and slight cough. Subnormal temperature.

November 21.—Patient still keeping quite well. No discharge from nostril, very slight cough. No albumen in the urine. Subnormal temperature.

In five other cases of which I have not the details, Dr. Harper used the antitoxin serum with marked success. Some of these cases were very bad. In one the membrane extended into the larynx, producing grave symptoms; in another, the only one examined, the Klebs-Loeffler bacillus was found.

Remarks.—Of these three detailed cases the two former were mild compared with the latter, but in all of them the improvement immediately succeeding the use of the Antitoxin Serum was so manifest that it was impossible not to attribute the recovery to its use.

Certain early appearances in the first two cases rather resembled follicular tonsillitis than diphtheria, but these rapidly passed into the conditions of the latter disease. The membranes which appeared, in their rapidity of growth, thickness and toughness, removed all doubt as to their real nature. Bacteriological examination in the first two cases did not take place, as the specimens sent to be thus examined were destroyed in transmission. In the third case the membrane was so examined, and the Klebs-Loeffler bacillus was detected.

Up to the present date there have been no signs of paralysis in either of them, but it is, perhaps, too early to say that that danger has passed. There have been many cases of follicular tonsillitis in Finchley, and a considerable number of cases of diphtheria.

In all probability the primary disease has been tonsillitis, and this has produced in the parts affected a condition of tissue which has afforded a favorable soil for the poison of the more serious disease.

Judging from the fact that in nearly all the cases of follicular tonsillitis that have occurred in Finchley, the milk supply has been from one source, the local authorities attribute the outbreak to this cause.

In the two former cases that I have related, the boys went to a school in Finchley, and there daily had a glass of milk; their residence was outside Finchley parish, and no other member in the same family was attacked, although the supply of milk at the house was from the same dairy.

The inference from these facts was, that if the milk was to blame, the cows from which the Finchley milk was taken were different from those from which the Hendon milk was taken.

On inquiry I found, from the milkman who delivered at the house, that this was so. But when I went and saw the manager at the dairy he was not so definite on this point, and implied that, at least sometimes, the milk from all the cows was mixed.

The drainage of the road which connects the Finchley and Hendon parishes along the portion of which these two boys had to go on their way to school is not satisfactory; there are openings in the road which appear not to be properly trapped, and which at times stink most offensively.

It is in this road, which is called Hendon Lane, that many of the houses stand in which the cases of throat mischief have appeared.

The school at which the three boys whose cases are related in full attended is situated in this lane.

There have been several boys absent from school, there have also been cases in the house itself, and the school has been closed.

The atmospheric conditions in the early days of November were also favorable to the production of follicular tonsillitis. The average percentage of moisture in the air for the first eighteen days was 81·2, and there was a steady fall of temperature, though not more than usual for the time of year.

In these three cases there seems to be ample opportunity for the occurrence of the disease from a possible combination of causes.

Dr. Klein, in his reports to the Local Government Board and elsewhere, has established the fact that diphtheria can be communicated by milk. He has, after poisoning the animals with the diphtheria bacillus, produced the disease in them, finding the bacillus in their organs after death, and been able to produce pure cultivations of the same microbe from milk drawn from them with all possible precautions against external infection.

Action of the antitoxin.—I can safely say I have never before witnessed a result from the action of a remedial substance so certain and so rapid. (The rapid action of the subcutaneous use of alkaloids approaches nearest to this result. But I do not know of any alkaloids which in ordinary medicinal doses produce anything like the organic change in a healthy direction which follows, and quickly follows, the use of the antitoxin serum.)

Within twenty-four hours a necrotic membranous exudation, which, since its recognition as a specific morbid condition, has more or less defied every remedy that has been applied, was visibly mitigated both in its growth and action, and diminished in its extent. In sixty hours—in the first two cases after the second injection—the membrane had entirely disappeared. Only one injection was used in the third case, the worst of the three, and this because no more of the remedy could be procured, and yet the membrane had disappeared seventy-two hours later.

I attempted to make out how the antitoxin acted on the diphtheritic membrane, and thought that I discerned an action of a somewhat solvent nature. In the cases under my care the membrane did not flake or shred off, but disappeared, becoming thinner, smaller in extent, and more translucent. In the third case a large piece is recorded as having been thrown off from the nostrils. Respiratory efforts may in this case have been instrumental in its removal. So far as my experience goes the antitoxin is not innocuous. Doubtless when the diphtheritic poison is present its action is expended thereupon. But in one case of follicular tonsillitis, in which I used it as a prophylactic, it produced very unpleasant results. A deep-coloured ring of confluent papules formed around the spot where it was injected, which in the course of the week spread all over the body, being accom-

panied by symptoms resembling those of rheumatic fever, and producing great prostration.

Dr. Roux, in the 'Annales de l'Institut Pasteur' for October, 1894, enunciates the view that antitoxin serum acts by stimulating the cellular elements of the system, rendering them not only insensible to the poison, but exciting phagocytic action in them destructive to the virus. This condition he points out, also, is only temporary. Permanent immunity from diphtheria he believes can only be obtained—as in the case of the horses from which this serum is obtained—by repeated and gradually increasing doses of the diphtheritic poison itself.

I think the exact dosage of the remedy requisite yet remains to be made out. Until this is so determined, it is best to be on the safe side, and give rather more than is needed than not to give enough.

I should be lacking in simple courtesy, to say the least, did I fail to take this opportunity of acknowledging my great personal obligation to Dr. Ruffer, who, from his then scanty store, amply and generously supplied my need of this remedy. I am also indebted to Mr. Butlin for his kind assistance and advice as to where to seek for the antitoxin, and to Dr. Harper, of Finchley, for the notes of his cases which he placed at my disposal.

XIV.—*Three cases of Abdominal Section for unusual conditions:*—(a) *Tubercular disease of the liver;* (b) *Complete volvulus and strangulation of the great omentum;* (c) *Traumatic hæmorrhage without external wound.* By A. W. Mayo Robson. *Read January* 11, 1895.

I MUST apologise for giving in the same paper the three following dissimilar cases, as they only resemble one another in the fact that they were all treated by abdominal section, which in each case resulted in complete recovery. I venture to hope, however, that each may be found to present points of interest sufficient to occupy the time of the Society for a few moments.

Case 1. *Tubercular abscess of the liver.*—G. R., æt. 31, a miner, was admitted to the infirmary on October 17, 1892, with the history of having been perfectly well up to twelve months before, when he commenced work in a damp mine, and immediately began to suffer from intermittent attacks of pain in the right knee and in the back. He was able to follow his occupation up to the 28th of March, 1892, when at 4.30 in the morning, while walking to his work, he felt a sharp pain in the epigastric region, which gradually increased in severity up to 12 o'clock, at that time becoming so severe as to necessitate his ceasing work. The pain was intensely agonising, causing him to roll about on the floor with the body doubled up. There was no accompanying vomiting or jaundice. There had been no recurrence of the intense pain, but a dull aching had been more or less constantly present, entirely preventing him working.

Three months prior to admission he noticed a lump at the seat of the pain; this had steadily increased in size. There had been a slight tendency to jaundice during the four months preceding admission, and epistaxis had occurred once or twice a week during the same period. His general health had failed, and he had lost half a stone in weight since June. No cough was present, but there were frequent night sweats.

When admitted under the care of Dr. Churton, the patient complained of a dull aching pain in the epigastrium, also high up between the shoulders, as well as in the left shoulder and

over the spine of the right scapula. To the right of the middle line in the upper epigastric region there was a smooth rounded tumour the size of an orange, apparently connected with the liver. Nothing abnormal was discovered in any of the other organs.

No improvement having occurred under medical treatment the patient was transferred to the surgical wards, and on October 22, 1892, the surface of the abdomen having been thoroughly cleansed, a vertical incision two inches long was made over the swelling just below the right costal margin. The liver was found adherent to the parietes, and the abscess cavity was opened as soon as the latter were cut through. A large quantity of pus and caseous matter was removed with a lithotomy scoop, and the space having been well washed out, iodoform was rubbed into its walls, a drainage-tube inserted, and the upper part of the wound closed. The stitches were removed a fortnight later, the upper part of the wound being firmly healed, and the tube two days later, a very small amount of discharge being present at each dressing.

A small sinus persisted for a time, from which a few drops of pus were discharged daily, but the patient's general health improved rapidly, and he was discharged on December 28. When seen in June, 1893, he was in good health, the fistula having healed, leaving a healthy scar.

The report of the Pathologist states that the material removed was shown by microscopic examination to be tubercular.

For the notes of the case I am indebted to my late house surgeon, Mr. A. L. Whitehead.

That the condition of tuberculosis of the liver as an independent affection is a rare one the following references would seem to prove; but that a radical treatment is promising is amply demonstrated by the present case, as I believe the patient is now in perfect health.

Taylor (*Manual of Medicine*, 2nd edit., p. 633) says tubercle of the liver is almost invariably a part of general tuberculosis.

Dr. Smith (*Quain's Dictionary*, art. "Liver, Morbid Growths of") repeats the same.

Dr. Roberts (*Theory of Medicine*, p. 714) says tubercle is usually found only in connection with acute miliary tuberculosis, but is occasionally secondary to chronic tubercular diseases elsewhere.

Osler (*Theory of Medicine*, p. 242) mentions that though most commonly found in acute miliary tuberculosis, still large caseating masses are occasionally found.

Pye-Smith (Fagge's *Principles of Medicine*, 3rd edit., p. 392) says that a large caseous tubercular mass, softening into a pseudo-abscess, is one of the rarest pathological curiosities, and he thinks that some of the few recorded cases are of actinomycotic origin.

Case 2. *Complete volvulus, with strangulation of omentum, producing acute peritonitis.*—R. B., æt. 35, was admitted to the infirmary on March 1, 1892, almost in a state of collapse from acute abdominal pain, the abdomen being distended and exquisitely tender. His pulse was 120 and intermittent. There was not complete obstruction, as he had passed flatus quite recently. He said that he had suffered from a left inguinal hernia for six years, and although he had worn a truss the rupture occasionally came down, but was easily reduced. Four days previously while doing some heavy work the rupture descended, and was reduced with a certain amount of force; but after reduction, which was unaccompanied with a gurgle, the abdomen was more painful than usual, and in fact he was so much distressed that he had to cease work, but his bowels were moved the same afternoon. The following day the pain had increased, but flatus passed freely, and there was no vomiting. On the third day vomiting came on, and persisted up to the day of admission.

On examining the abdomen little could be made out on account of the great tenderness and distension. On passing the finger, by invaginating the scrotum, up the left inguinal canal, which was very patent, a hard tender mass could be touched within the internal ring.

After a consultation of the surgical staff, who were all present at the time of operation, the abdomen was opened in the middle line below the umbilicus, when it was discovered that the great omentum, attached below to the inguinal canal by a cord about the thickness of a penholder, and above to the lower border of the stomach by a similar cord, had completely revolved several times on its axis, thus forming a large hard mass, which was passing from a state of congestion to one of strangulation. The two cords above and below presented several twists, and after division of these the mass of omentum was removed.

A quantity of sanious peritoneal fluid was removed by

sponges, and the peritoneum closed after ligaturing the aforementioned pedicles,—not because they were bleeding, but as a precautionary measure.

After removing the mass it was found to consist of the whole of the omentum except the small fan-shaped portion coming down from the stomach to the upper twisted cord.

After the operation there is nothing special to record. The wound healed by first intention, and the patient was allowed to be up on the 20th of March, and was discharged on the 26th.

So far as I know the case is unique, and I think that in all probability a correct diagnosis was impossible. The whole staff agreed with me in thinking there was strangulation of omentum in a hernial sac which had been reduced *en masse*, the free passage of flatus clearly indicating that the bowel was free from strangulation.

It clearly demonstrates the importance of operating in doubtful cases of peritonitis, although the cause may be undiagnosed, as indeed it is frequently undiagnosable.

Case 3. *Traumatic intra-peritoneal hæmorrhage treated by laparotomy with lavage and drainage.*—On July 18 the patient, James H., æt. 13, was attacked by a bull in the cattle market. The beast pushed him against some railings and fixed him there, its face being in contact with the boy's abdomen, and its horns on either side of him. When released he felt sickly and had abdominal pain, but walked home, a distance of half a mile, without help. His friends put him to bed, but the abdominal pain continued, and he began to vomit. Late in the evening of the same day he was admitted to the Leeds General Infirmary. He looked very ill, face pinched and pale, thighs flexed on abdomen, pulse 130, temp. 100°. The abdomen was fairly full, with dulness in both flanks; there was slight general tenderness. He passed a little urine full of urates. Liver dulness present. There was bruising of the back in the lumbar region, extending outwards to the left loin.

Next morning his temperature was normal, the pain continued, the abdomen was much fuller, and the dulness more extensive in the flanks, now extending into the iliac and hypogastric regions; fluid thrill; pulse 128, small and feeble. The friends were telegraphed to for permission to operate, as the patient was rapidly getting worse, and there was mani-

festly internal hæmorrhage. Early in the afternoon the abdomen was opened by an incision 1½ inches long in the linea alba midway between the umbilicus and pubes. Bright red fluid blood gushed out, of which two pints were measured. The peritoneal cavity was flushed with about ten pints of hot saline solution, but his pulse became so rapid and feeble, in fact almost imperceptible, that it was decided to trust to drainage to stop the bleeding, as it was manifest that if the operation were prolonged the patient would die on the table; hence, although the lotion returned from the abdomen deeply stained with blood, a glass tube was put in to drain the pelvis, and the wound was closed. When he left the theatre the pulse was 150. Half an hour after, 2 ounces of deeply stained fluid were removed through the tube. At the second dressing, an hour later, 1 ounce of similar fluid, and at the third dressing, two hours later, only ʒij, and four hours later again ʒij.

Next morning, July 20, at 9 A.M., ʒij of clearer fluid were withdrawn, and at 12 noon ʒj was removed and the tube taken out.

His temperature was 98° and his pulse 112, and although he looked ill and pale, he was comfortable and cheerful. He had passed urine, and the abdomen was quite flat. The case progressed very favorably, and he got up on August 9, and went home on August 11, walking to the front door with ease.

On September 8 he was seen at "out-patients," looking very well.

The case illustrates very forcibly the hæmostatic effect of washing out with hot solution combined with drainage, and demonstrates the fact that in some cases of intra-peritoneal hæmorrhage where the patient is too ill to bear a prolonged operation this simple method may still save life by first emptying and then keeping the peritoneal cavity emptied, thus allowing coagulation to take place in the opened vessels.

XV.—*Loculated Empyema—a case for diagnosis.* By J. W. Browne, M.D. *Read January* 11, 1895.

R. G. B., æt. 46, a strong, healthy-looking man, with a good family history, was first seen on September 25, 1891. His personal history was this:—Some time between eighteen and twenty-five years of age he had syphilis, so lightly that he recollects no more than the fact. At the age of twenty-three, after five years in the P. and O. service, he was invalided, and finally discharged for "disease of tropical climate," whose symptoms were emaciation, weakness, and an acute pain along left lower ribs; when reappearing three years later it was called "spinal neuralgia." About 1889 he had very severe pain in the back, which ended in a considerable discharge of pus *per urethram*: a specialist treated him for gonorrhœa, but his medical attendant was convinced that the pus came from one of the kidneys; this fact came to light only lately and indirectly. In 1888 he was insured as a first-class life. In 1890, about a year after marriage, his wife bore a healthy child, and another in 1892, which, however, suffered from obstinate sores on the nates. Early in 1891 he had a sharp attack of influenza, with a slow recovery; since—*post*, perhaps *propter*—he has suffered from pain and weakness in the lower limbs, especially the right, after prolonged exertion; this symptom he called sciatica, and attributed to a strain at lawn tennis.

When first seen he complained of a pain in the stomach, to which he had been subject for two years; it was often associated with vomiting; its seat and character suggested a diagnosis of gall-stones. In the course of the next few days, however, no jaundice supervened, the pain spread to the back, grew much worse, and was even agonising on deep inspiration or the slightest movement; there were profuse sweats, irregular fever below 100° F., and finally marked signs of right pleurisy with effusion reaching to about the angle of the scapula. He was seen in consultation September 30, and the diagnosis made of pus or serum in the right pleura, most probably of hepatic origin. During the next seven days, however, the signs of fluid became much less marked. The fever, the sweats, and the constitutional disturbance passed away, and he was apparently convalescent

save for some slight and occasional pain, which seemed now to be centred under the xiphoid. Here, on the evening of October 7, the patient felt as though something had burst inside, followed by excruciating pain and symptoms of collapse. Opiates and stimulants aided him through the night; next morning the only objective change noticed was a very evident displacement of the heart's impulse to above and rather outside the left nipple, where, too, could be seen a diffused undulatory movement. The abdominal tenderness, greatest at the xiphoid, was diffused, especially over the line of the ascending colon. The same day an exploratory incision was made antiseptically from the xiphoid in mid-abdominal line for two or three inches, but nothing abnormal was seen or felt to account for symptoms. At the same time a hypodermic syringe was plunged into the eighth space of the right back, and again lower down; only blood came. Soon after the operation there was deep jaundice, and jaundice was for months an irregular symptom, associated invariably with pain, located now over the gall-bladder, and again under the xiphoid. The wound healed speedily. As the abdomen became flaccid a band of tympanitic resonance was noticed, which, extending from just below level of left nipple to xiphoid level, where it merged in abdominal resonance, stretched across from left axillary line to about an inch beyond the sternum. On October 15 the pain and friction of pleurisy were observed below left nipple, spreading to the back, and followed by impairment of resonance, breath-sounds, and fremitus.

A diagnosis was now made of hepatic or vertebral disease causing right and then left pleurisy; the displacement of the heart was attributed to fluid in the right pleura, and right paracentesis was urged and performed without result. From October 10 to December 8 there were cough, irregular fever, sweats, steady loss of flesh and strength, occasional attacks of alarming collapse, and pain suggestive now of left pleurisy, now of left peritonitis; sometimes the patient complained of chilliness, but there was never at this stage any frank rigor.

The state of his chest was such as to suggest the suspicion on October 28 of the escape of pus from pleura into left lung, and of pericarditis: on October 31 of limited left pneumothorax with consolidation of lung: on November 21 of right and then left pleurisy—with gall-stones as an independent and unassociated accident: on December 1 of collapse of lung and consolidation, possibly tubercular or syphilitic, of left

base. After these differences of opinion the physical signs at last became fairly constant; at the right base of old pleurisy, now quiescent; on the left side the heart's impulse was felt an inch above and in the line of the left nipple, at the left apex exaggerated breath-sounds and resonance down to the fourth space, below that tympanitic resonance; over the left back resonance, fremitus, and breath-sounds were everywhere impaired, nowhere abolished, most impaired just below left scapula; everywhere adventitious sounds, coarse and fine, as of pleura, lung, or both.

Finally, on December 6, an exploring needle and syringe were plunged into the maximum of dulness, apparently with no result; afterwards, on washing the needle, a drop of stinking matter was discovered. The next day a surgeon made a free incision with resection of ribs into what proved to be a loculated empyema, which was well drained of its foul contents. In two months the patient was up and out, rapidly regaining flesh and strength. But for some months afterwards he had attacks of what seemed to be biliary colic with rigors, profuse sweats, jaundice, and clayey stools. His motions were repeatedly searched, and once only were found two small black pills, yellow inside, which an authority pronounced to be probably gall-stones. About this time suggestions were made of hydatids of the liver and of pus in the right pleura. At any rate, the man is about his business as well as ever. His stomach is still displaced, and probably adherent. His heart is displaced; there is a little dulness at the right base, and that is all. But he still complains of the pain and weakness in his lower limbs, which were in abeyance during his long illness. He has no appreciable loss of muscular power, no inco-ordination, no paræsthesia; the knee-jerks are rather exaggerated; his spinal column seems normal.

The interest of this persistent symptom lies in its support of a probable diagnosis of spinal caries—of unknown cause—as the explanation of the whole case. Gall-stones, no doubt, he had—and subsequent ulceration of the gall-passages with pyæmia might account for all the symptoms of his latest illness; but this diagnosis would not account, as caries of the vertebræ would and could, for the spinal neuralgia of his youth, for the later discharge of pus *per urethram*, and the present weakness of the lower limbs.

XVI.—*Case of Acute Intestinal Obstruction from Gall-stone: laparotomy: removal of stone: suture: recovery:* with a table of previous cases. By Frederic Eve. *Read January* 11, 1895.

GEORGE S., engineer, æt. 42, was admitted to the London Hospital on March 19 at 5 P.M., presenting symptoms of acute intestinal obstruction.

History.—Four days previously (March 15) he was attacked with pain in the abdomen, and immediately afterwards vomiting commenced. From that time he had been unable to keep down any food. There had been passage of neither fæces nor flatus since the onset of the attack. He was questioned as to his having had any previous attacks of obstruction or any attacks of inflammation about the abdomen, but no information on these points could be elicited. Subsequently, after the operation, he recalled that eighteen years previously he became subject to severe attacks of pain with tenderness in the epigastrium; they were accompanied for the first six years with vomiting. For seven or eight years he was never free from pain, and about once a week had a severe attack. In 1884 he was suffering so greatly that he consulted a well-known physician, who ascribed the pain to dyspepsia. Six months after this the persistent pain, together with the acute attacks, ceased suddenly, and he had no recurrence. His bowels were always constipated, but he usually had a daily evacuation. He had never passed a gall-stone.

I saw the patient immediately after his admission; the abdomen was moderately distended, but no distension of the large intestine could be made out; the pain was referred to the umbilicus: no tenderness could be elicited. No vomiting after his admission. The hernial apertures and rectum were free. A large injection of soap and water (about two quarts) was given, the patient being inverted as far as possible with pillows placed under the hips; tincture of belladonna, 15 minims, was ordered every two hours. About 8 P.M., after the return of the enema, he passed a small fluid motion (about 4 oz.) and felt easier. The operation was in consequence postponed. Next morning his symptoms became more severe: he had passed nothing further by the bowel, and laparotomy was therefore determined on.

Operation at 1.15 P.M.; anæsthetic, ether; on palpation of the abdomen, when the muscles were relaxed under anæsthesia, a hard body like a rifle bullet could be felt in the right iliac fossa. This was evidently a gall-stone. On opening the abdomen the gall-stone was readily seized; it lay in the lower end of the ileum two or three inches from the ileo-cæcal valve. The intestine above the obstruction was considerably distended, and of a rosy-red colour but glistening; there was no evidence of peritonitis. The large intestine was completely empty. The ileo-cæcal orifice was exposed to view by traction on the wound and intestine respectively, and an attempt was made to force the stone through the valve into the cæcum, but unsuccessfully. The calculus was quite moveable, and was pushed along into a free knuckle of ileum, which could be readily drawn out of the wound. After applying elastic ligatures of drainage-tube to the intestine above and below, the calculus was removed by a longitudinal incision. The mucous membrane was then united by a continuous suture of silk, and the serous covering by interrupted Lembert sutures, also of fine silk. A free end of omentum was then attached by four sutures over the wound, and the intestine was dropped into the abdomen and the wound closed. The subsequent progress of the case was uneventful. There was no recurrence of the vomiting, but the patient suffered during the next night from hiccough. On the third day the bowels acted copiously twice, and there was no further trouble in that respect. On the evening of the first day his temperature rose to 101·4°, but immediately fell and remained normal until the ninth day, when it again rose to 101°. The wound appeared in the meantime to have closed by first intention, but on the tenth day there was a spontaneous discharge of fœtid pus from its lower angle, and it was then found that the skin had been undermined by an abscess, either due to a suture, or possibly to contusion of the edges of the wound by the retractors. There was never any suspicion of fæcal fistula, and union subsequently took place without further complication. Nutrition was maintained entirely by the rectum until the fourth day, when a little Valentine's meat juice was allowed, as his strength seemed to be failing. The rectal alimentation was continued for seven days, the amount of food given by the month being gradually increased. He left the hospital feeling quite well in three or four weeks.

Remarks.—Before the patient was anæsthetised the diagnosis of obstruction of the small intestine was made, but no

clue as to its cause could be obtained. The sudden onset of pain immediately followed by vomiting pointed to internal strangulation, but, considering that four days had elapsed since the onset, the general symptoms did not seem sufficiently acute to support this view: as already mentioned, inquiry at that time elicited no information as regards his previous attacks of abdominal pain. The distension of the abdomen was too great to allow the stone to be felt before he was anæsthetised. The long antecedent attacks of epigastric pain may be referred to the presence of the calculus in the gall-bladder, culminating in the establishment of a fistulous communication with the duodenum; while the sudden cessation of the symptoms after existing some seven or eight years was probably concurrent with the passage of the calculus into the small intestine. The condition of the calculus warrants the assumption that it had remained for a long period in the intestine, for, except at one end, it is covered with a distinct layer of phosphates. (In Mr. T. Smith's successful case fifteen years had elapsed from the entrance of the gall-stone into the intestine to the occurrence of obstruction.) The attack of obstruction in the case related may have been determined by the calculus being driven against the ileo-cæcal valve, and consequent inflammatory swelling of the mucous membrane. The suggestion of Mr. Lawson Tait to break it up with a needle occurred to me before extracting it; but I was deterred from doing so by its feeling of hardness, and by the fact that I could readily guide it to a healthy and only moderately distended portion of the bowel, which I thought would readily unite after incision and suture.

The calculus is cylindrical in shape, rounded and smooth at one end, broken and somewhat rough at the other. It appears to be composed chiefly of bile-pigments, biliary acids, and salts, and is covered with phosphates. It measures 1½ inches (4 cm.) in length, and over 3¼ inches (8·5 cm.) in its greatest circumference, and weighs, dry, 190 grains. The calculus was found in the usual situation: thus, of thirty-two cases collected by Leichtenstern, it was found ten times in the duodenum and jejunem, five times in the middle of the ileum, and seventeen times at the lower end of the ileum.

So much has been written regarding the symptoms of obstruction from biliary calculus that I do not think any useful purpose would be served by going into this matter. Suffice it to say that in the absence of the history of previous

pain and inflammation in the regions of the right hypochondrium and epigastrium, I do not think a diagnosis was possible in the case above related, except by palpation under an anæsthetic.

The results of the treatment of this form of obstruction by laparotomy were at first by no means encouraging. Writing in 1889,* Dufourt collected nine cases in which this operation had been performed. All of these terminated fatally with the exception of one, that published in the 'Transactions of the Clinical Society' (vol. xxi, 1888) by Mr. Clutton. In this case the calculus was pushed into the large intestine. In two cases the calculus was not found at the operation, but its presence was subsequently verified *post-mortem;* and in the six other fatal cases the intestine was opened.

In the period subsequent to 1889 I have collected sixteen cases including that just related, together with two cases published in 1887, and overlooked by Dufourt; among these there were nine cures and nine deaths. In all of these cases (except one in which resection was performed) the bowel was opened and sutured. This shows a very great advance, which may be attributed mainly to improved surgical technique. Operation was no longer delayed in the earlier than the recent cases; for, excluding one fatal case in which operation was postponed sixteen days, the average for both groups was four days from the onset of symptoms. The improvement in the recent cases is still more apparent if we exclude from the nine fatal cases two in which the unfortunate result could not be attributed to the operation, and one in which resection of 20 centimetres of the intestine was necessary. In the latter case, reported by Bircher, the knuckle of intestine containing the calculus showed in the neighbourhood of the mesentery some yellow points, and gave out a fæcal odour. The mucous membrane at this part was covered with a number of ulcers, the largest being 7 centimetres in length and 2 in breadth.†

Regarding the two cases in which the fatal result was unconnected with the operation, in one, that of Helferich, there existed between the perforated gall-bladder and the duodenum a little canal in which one half of the calculus was retained, the other half having passed into the intestine and determined the obstruction. The migration of the latter half of the calculus had caused a local peritonitis in the neighbour-

* *Lyon Médicale*, 1889, p. 53.

† *Revue des Sciences médicales*, vol. i, tome xli, p. 278.

hood of the gall-bladder, resulting in paralysis of the intestine, to which the author attributed the fatal result. In the second case, in which Von Winiwater was the operator, a woman aged eighty had symptoms of complete obstruction for twenty-four hours; there was an irreducible right femoral hernia; herniotomy was performed, and the knuckle of intestine (showing a depression, but not apparently strangulated) was returned into the abdomen. Next day, the symptoms being unrelieved, laparotomy was performed, and a gall-stone found and extracted from the intestine just above the knuckle, which had occupied the hernia. As the latter looked suspicious, the intestine was fixed to the wound and a tampon of iodoform gauze applied around it. Perforation of the intestine at the seat of the constriction took place, and the patient died with delirium and exhaustion.

Excluding, therefore, these three from the recently recorded cases, we have nine cures and six deaths out of a total of fifteen.

One of the earlier cases in Dufourt's list was said to have died from persistence of the obstruction, occasioned by a contraction of the large intestine at the end of the transverse colon (Labbé, *Thèse de Beraud*, 1885).

Taking the earlier and later cases together there are twenty-seven in all, of which seventeen died and ten survived the operation of laparotomy.

It must, however, be borne in mind that doubtless several unsuccessful cases have not been placed on record.

It is, perhaps, scarcely too much to say that as a result of this inquiry the operation of laparotomy, with extraction of the calculus by incision, is placed on a much more favorable basis than it has heretofore occupied. To sum up, the following proposals regarding treatment may be formulated.

As in the past, so in the future, it is probable that in the majority of cases the cause of the obstruction will not be discovered until after the abdomen has been opened. But when a diagnosis of obstruction by a biliary calculus is possible, expectant treatment (namely, the administration of belladonna or opium and rectal feeding) is indicated if considerable distension of the abdomen is not present. Treves* states that of twenty cases in which gall-stones "produced definite and severe symptoms of obstruction" six patients recovered by the spontaneous passage of the stone, and fourteen died unrelieved. Should the measures mentioned

* *Intestinal Obstruction*, p. 335.

fail, operation should not be long delayed, as the patients are nearly all old people whose vitality will soon give out. Of twenty cases operated upon in which the age is stated the mean age is sixty-four years. By far the youngest was my own patient, aged forty-two.

After opening the abdomen the procedure to be followed must depend on circumstances.

If the stone occupies the lower part of the ileum and is moveable, an attempt should be made to push it through the ileo-cæcal valve. This proceeding is devoid of danger, for in only one case, that recorded by Körte, has a biliary calculus become impacted in the large intestine and given rise to obstruction. The site of the impaction in this case was the sigmoid flexure, and extraction met with a favorable result, although a fæcal fistula formed.

Should it be impossible to pass the stone through the ileo-cæcal orifice, or if the stone be immoveable, as is not infrequently the case, the procedure to be adopted should depend on the condition of the intestine. If the intestine be healthy and easily reached from the wound I would advise incision and suture as in the case related. But in those cases in which the intestine is inflamed and œdematous, the stone fixed and adherent, the procedure recommended by Mr. Lawson Tait, namely, breaking up the calculus with a needle, would appear to offer the best chance of a favorable result. Again, should this proceeding be impracticable, I would advise the method adopted in von Winiwater's case (already alluded to); that is, to excise the stone and suture the intestine. Then fix the intestine to the abdominal wound, surround the wounded part with iodoform gauze, and plug the wound with the same, subsequently dealing with the case as might be found necessary.

In cases similar to that recorded by Bircher, where ulceration of the intestinal wall is manifestly present and perforation is threatened, there is no other resource but resection and union of the intestine by Murphy's button, or the formation of an artificial anus.

When a stone has been extracted by incision the opportunity of evacuating the intestinal contents through the opening should not be lost.

Name.	Age and sex.	Operation.	Result.	Duration of symptoms.	Seat of stone.	Peculiarities.	Cause of death.	Reference.
T. Smith and Dr. Ford Anderson	M., 65	Incision and suture of intestine	R.	Obstruction six days	...	Gall-stone entered bowel 15 years previously	...	Lancet, Dec. 3, 1887.
Thiriar	...	Do.	R.	...	End of small intestine	Stone weighed 14 grammes	...	Traité de Chirurgie, Duplay and Reclus, vol. vi, p. 423.
Sick	F., 64	Do.	R.	6 days	Middle of small intestine; almost immoveable	Stone felt before operation in left inguinal region	...	Deut. med. Wochenschrift, 1891, No. 7, s. 268.
Rehn	...	Do.	R.	...	Middle of small intestine close to valve	Stone felt through abdominal wall	...	Verhandl. Deut. Gesell. f. Chir., 1889, s. 107.
Helferich	F., 67	Do.	D.	Ileus 5 days	1 metre above valve; obstruction caused by one half of stone; other in fistula	...	Peritonitis about fistula between gall-bladder and duodenum 43 hours after operation	Ibid., s. 110.
Von Winiwarter	F., 80	Do.	D.	Several days acute ileus	...	Irreducible right femoral hernia; hernia opened; intestine and omentum returned; stone lodged in intestine above hernia	Perforation of bowel at seat of strangulation. Patient died a few days after operation of delirium and exhaustion	Beiträge f. Chirurgie, 1892, s. 511.

Name.	Age and sex.	Operation.	Result.	Duration of symptoms.	Seat of stone.	Peculiarities.	Cause of death.	Reference.
R. Nairn	F., 75	Incision and suture of intestine	D.	Obstruction 6 days	Jejunum	Stone 3 inches in circumference	10 hours after operation	Trans. Clin. Soc., vol. xxv, p. 211.
Bruce Clarke: Case I	...	Incision and suture	R.	...	Quickly found	...	...	...
„ II	F., old	Do.	D.	...	Not easily found	...	Sank	Lancet, May 20, 1893.
Mr. Mercer	F., 58	Do.	D.	Ileus	...	...	Died next day	...
W. Körte: Case I	F., 71	Do.	D.	Ileus 2 days	...	Small femoral hernia opened	Died from collapse in 12 hours; no peritonitis; sutures secure	Arch. f. klin. Chir., Bd. xlvi, Heft 2, 1893, s. 331.
„ II	F., 66	Do.	R.	Ileus 6 days	Half metre above valve	Stone fast fixed	...	ditto
„ III	M., 76	...	R. Fistula which soon closed	Ileus 3 days	Sigmoid flexure; not moveable	Crural hernia explored; nothing found	...	ditto
Bircher	F., 60	Excision 20 centimetres of intestine; enterorrhaphy in three stages	D.	Ileus	...	Intestines showed yellow points and gave out fæcal odour; mucous membrane ulcerated	Collapse 10 hours after operation	Revue des Sciences méd., t. xl, p. 278.
Pouzet	F., 75	Incision and suture	R.	Ileus 5 days	Not noted; stone mobile	...	...	Ibid., p. 279.
B. H. Allen. Mr. Beck operated	F., 67	Do.	D.	Obstruction 16 days	Stone immoveable; seat not mentioned	Attack of colic 10 weeks before	25 hours after operation	Lancet, April 30, 1887.

Pearce Gould	F.	Do.	D.	...	Ileum	Umbilical hernia explored; nothing found	29 hours after operation	Ibid., May 20, 1893.
T. Bryant	F., 50	Do.	D.	72 hours	1 foot above ileo-cæcal valve	...	Peritonitis 8 hours after operation	Ibid., March 8, 1879; quoted by Dufourt.
Whalters	F., 56	Do.	D.	7 days	...	...	Recent peritonitis in 24 hours	Ibid., 1881; quoted by Dufourt.
Labbé	F., 56	Do.	D.	4 days	...	...	Persistence of obstruction due to narrowing of large intestine on 3rd day	Do.
Taylor	M., 66	Do.	D.	36 hours	...	...	No peritonitis	...
Hugh R. Ker	...	...	D.	36 hours	Stone just above valve, immoveable	...	Death in 4 days; no autopsy	Lancet, Nov. 26, 1881.
Brookhouse	F., 56	Do.	D.	8 days	...	...	Gangrene of wound and septicæmia	Quoted by Dufourt.
Clutton	F., 70	Calculus pushed into large intestine	R.	24 hours	8 inches above valve	...	...	Quoted by Dufourt; and Trans. Clin. Soc., vol. xxi, 1888.
Ord	...	Laparotomy; calculus not found	D.	...	...	...	...	Quoted by Dufourt.
Williams	F., 60 to 70	Do.	D.	...	...	...	...	Do.
F. Eve	M., 42	Incision and suture of intestine	R.	5 days	Just above valve	...	...	Case related above.

XVII.—*Two cases of Detachment of the Retina in the course of granular kidney.* By Samuel West, M.D. *Read January* 25, 1895.

CASE 1. *Granular kidney; rapid loss of sight; purpura; retro-bulbar hæmorrhage; detachment of the retina (not hæmorrhagic).*—Lydia B., æt. 22, was admitted into St. Bartholomew's Hospital under my care on March 2, 1893, suffering from headache, vomiting, and failure of sight.

She stated that she had been in her usual health until nine months ago, when the present symptoms began. Since then she had got gradually worse and worse, until ten days ago she became quite blind.

It was ascertained that she was an in-patient in Faith Ward seven years before, and that her case was diagnosed as chronic nephritis of four months' duration. The following account is given of her case at that time. She had considerable dropsy of the face and body, and passed but little water, with about 6 per cent. of albumen. A few days after her admission she had several fits, sixteen in one day, and they were followed by noisy delirium, ending in incomplete coma. At that time she lost her sight temporarily, but there were no eye lesions discovered by the ophthalmoscope, so that the attack was thought to be one of uræmia with uræmic amblyopia. She seemed likely to die for some days, but she rallied and rapidly improved, so that she left the hospital after a stay of nine weeks "wonderfully better." The albumen on leaving was reduced to 1·6 per cent.

Soon after leaving the dropsy reappeared, and disappeared again, but since then she has had several slight attacks of the same kind, so that she had been always more or less ailing until nine months ago, when the sickness, vomiting, and failure of sight set in for which she sought admission now.

Nocturnal micturition had been constant with her, at any rate since the illness first described, but it is not clear that it did not precede it also; but she had only recently noticed any marked increase in the amount of urine, and in the frequency of micturition by day. No history of scarlet fever could be obtained. The family history was negative, her father, mother, her two brothers, and one sister being all alive and well, and one sister only having died as an infant.

Condition on admission.—A fairly nourished woman, very pallid, with a little œdema of the eyelids, feet, and lumbar region. The breath somewhat ammoniacal. Respirations quiet and lungs normal. The tongue clean and moist. Appetite good and bowels regular. The pulse was 80, of increased tension; the artery thickened. The heart's dulness increased slightly upwards and a little towards the right side; the impulse forcible; the first sound prolonged at the apex and reduplicated; the second sound at the base accentuated; no murmur. The urine contained one third albumen; no casts were found. The headache was almost constant, and at times very bad all over the head. The vomiting did not bear any relation to the taking of food, and was very irregular in occurrence. She slept badly, chiefly from restlessness. Ophthalmoscopic examination revealed extreme double albuminuric retinitis, with numerous white patches, especially round the yellow spot, and hæmorrhages. The discs were considerably swollen. Vision was limited to perception of light only. The conjunctivæ were a little glassy and swollen from slight œdema.

Six days after admission (March 15) several small purpuric spots were observed on the body, legs, and arms, and over the buttocks a few large purple patches of extravasation developed. These gradually faded and disappeared, the headache and vomiting became less, the breath less ammoniacal, the œdema almost disappeared, and altogether the patient seemed better.

On March 19 detachment was discovered in both retinæ. This could not have developed more than two or perhaps three days before it was detected, for frequent examinations of the eye were made by myself and by my house physician. The detachment was peripheral, and at first was limited to the south or lowest part of the retina. It rapidly spread, and in a few days involved the whole periphery except the middle upper segment. No change had occurred in the patient's symptoms to indicate any fresh complication in the eye, and the only new symptom was the occurrence of moderate and repeated epistaxis for a day or two before the detachment was discovered. The colour of the detached portion of the retina was dull grey, and no sign of hæmorrhage was found then or afterwards. No change took place in the detachment after it had once fully developed, either by way of extension or diminution, nor did it ever spread to within a fair distance of the disc.

In other respects the patient seemed to be improving; the pallor was less extreme, the œdema had disappeared altogether, the albumen had diminished to little more than a cloud, the headache was better, and the vomiting had almost ceased. She was taking her food well, and slept soundly. The urine varied in amount a good deal day by day, but averaged about 50 ounces; the patient seemed to be going on fairly well until April 9, when she was suddenly seized with severe pain in the left eye attended with flashes of light. The eye became protruded somewhat, and the conjunctiva as well as the eyelids and adjacent part of the face became swollen. She also vomited several times. There was no rise of temperature or other change.

The question arose whether she had hæmorrhage into the eye (hæmorrhagic glaucoma) or behind the orbit. On account of the swelling of the external parts it appeared to me to be retro-bulbar, and this it ultimately proved certainly to be; for the next day the conjunctiva became ecchymosed, and when the eye could be again examined the retinal condition was found unchanged, and there was no trace of hæmorrhage within the bulb. The pain rapidly subsided under treatment, and the general condition of the patient seemed to be in no wise changed.

On April 19, during the night, the patient became suddenly quite delirious, and unable to recognise anyone about her; she complained of pins and needles all over the body, and had some general twitchings. Under cannabis indica and one extra dose of pilocarpine she was quieted, and the next day she seemed to have recovered.

May 15.—There is little to record; she remained *in statu quo*. Perhaps there was a little general improvement in health, but none in the condition of her eye. Finally she became tired of the hospital and went home.

The treatment consisted of perchloride of iron three times daily, and nitrate of pilocarpine gr. $\frac{1}{12}$, subsequently increased to $\frac{1}{8}$, night and morning, with an occasional dose of extract of cannabis indica to secure sleep.

Case 2. *Granular kidney; sudden loss of sight; detachment of the retina; death; granular kidneys found.*—Frederick F. S. was admitted into John Ward in St. Bartholomew's Hospital on March 2, 1893, under my care; complaining also of vomiting, headache, and loss of sight.

He stated that he was in good health and at work until

two months before his admission, when he began to vomit every morning, and to be much troubled with headache. It was not till February 24, or six days before admission, that he first noticed that his eyes ached, and that things looked misty. Up to that time his eyesight had been good.

He gave absolutely no history of illness prior to two months ago, except that he had a slight attack of congestion of the lungs nine years before, from which he quite recovered. He had never had scarlet fever so far as he knew.

His mother and two brothers were alive and well, but his father was dead.

On cross-examination he admitted that he had to get up at night for some years past to make water, and he had lately passed more urine than usual and had been thirsty, but with this exception he seemed to have been free from symptoms of any kind till two months ago.

He was a pale, anæmic man, with a somewhat sallow, earthy complexion. Tongue flabby and moist; appetite fair; bowels regular. Breathing good and lungs natural. Pulse 84, regular, of high tension. Artery much thickened and tortuous. Heart-dulness a little enlarged. The first sound at the apex prolonged and booming, and the second at the base accentuated. A systolic murmur (hæmic) was heard at both bases, but it varied greatly and was sometimes absent. The urine was acid, 1010, contained one-fourth albumen, and gave a slight blood reaction. Some finely granular casts were stated to be present, but I did not see them myself, and they were not found again.

There was no œdema of any part of the body, but the eyelids looked a little puffy.

Ophthalmoscopic examination revealed well-marked albuminuric retinitis. The discs were hazy and somewhat ill-defined, but there was no swelling or effusion. There were numerous white patches, and a few recent and small hæmorrhages. Vision was almost completely lost; all that could be seen were large objects placed near to eyes against the light.

The urine was much increased in quantity, and was on several days more than 100 ounces, and only rarely less than 60 ounces.

Except on the first occasion (?) no casts were found. The amount of albumen continued fairly constant throughout at about 2 per cent.

On account of the condition of the eyes and the headache three leeches were applied to each mastoid, and a small blister

to each temple, with some relief to the headache. No other improvement occurred, and on the 18th the retinal hæmorrhages had greatly increased.

On the 19th detachment of the retina was discovered on the right eye, the left being unchanged. The detachment was on the nasal side, and involved about one third of the periphery. By the 21st it had extended so as to involve the whole periphery except the N.W. segment. Nothing had occurred in the condition of the patient to suggest any fresh complication on the side of the eye, and the only fresh symptom was an attack of troublesome but not copious epistaxis at the same time that the hæmorrhages were found to be increased in both eyes (*i. e.* on the 18th), but the day before the detachment occurred.

From this time the patient rapidly lost ground, becoming weaker and weaker every day. On the 28th troublesome diarrhœa set in, which still further reduced him, and on the 30th epistaxis recurred, which had to be treated by injection of perchloride of iron.

On the 1st of April he died, having during the last three days of life been greatly troubled with vomiting, which caused him to reject and refuse all food, and having passed but little water—about 25 ounces daily. He died quietly of asthenia, and was conscious to the last.

The patient was treated throughout with nitrate of pilocarpine, gr. $\frac{1}{8}$, three times daily by the mouth.

Remarks.—Granular kidney is a disease which long lies latent, *i. e.* it is only after it has existed for some time that marked symptoms arise. When they once appear they often develop with very great rapidity. This fact may be read the converse way, and we may say that when granular kidney produces symptoms it must have existed for some time. Now this is an important fact, for it has to be reckoned with in determining the ætiology of the disease. It sometimes happens that the first symptoms of granular kidney are œdema and other signs of acute nephritis. If the case runs an ordinary course the patient will recover up to a certain point, but never entirely lose the albumen in the urine. Whence the conclusion might be drawn that the granular kidney ultimately found resulted from the attack of acute nephritis. Yet the opposite conclusion would be the correct one, viz. that the acute nephritis had developed because the kidneys were previously diseased. The constant difficulty in acute nephritis is to determine whether it is really a fresh affection

or merely an intercurrent exacerbation of a chronic disease. I have seen many such cases. In some of these albuminuric retinitis has been present. That to my mind is practically conclusive, for I have never seen albuminuric retinitis in simple acute nephritis.

Suddenness of onset of symptoms characterises granular kidney throughout. Thus in one patient the first symptom may be the signs of acute nephritis as described, in another uræmia, in another hæmorrhage, *e. g.* violent epistaxis or even intra-cranial hæmorrhage; and in another—and this is of chief interest in respect of the present cases—sudden loss of sight. And we may expand this fact still further, for not only may serious symptoms arise in granular kidney without warning, but when they have developed they may be taken as an indication that the case is nearing its end.

Loss of sight.—In both these cases the failure of sight was rapid. It is true that in the first case there had been some gradual failure of sight for about nine months, but in a few days the patient suddenly became blind—totally blind, so as to have lost all except mere perception of light. In the second case no defect of vision was observed until six days before the patient became totally blind.

I was asked some time ago to see a gentleman in Essex who had lost his sight suddenly, and had become very ill. I found him evidently bordering on uræmia, with extreme albuminuric retinitis. He was obviously the victim of granular kidney. Pericarditis developed in a few days, which carried him off. Yet three weeks before his death he was out hunting, and the first thing he noticed was that he could not see a wire fence, and got into difficulties in consequence. Within a fortnight from that time he had lost all vision except the perception of light, and he died, as I have said, with pericarditis. This sudden failure of vision in granular kidney may be read the converse way, viz. that albuminuric retinitis generally exists for a long time without causing any symptoms, so that its presence will be only discovered by routine ophthalmoscopic examination.

Hæmorrhage.—The next point I desire to refer to in relation to these cases is hæmorrhage. The wide-spread morbid changes in the vessels throughout the body incline me to the opinion of those who regard granular kidney as a disease *sui generis*, and not primarily an affection of the kidneys at all; the disease of the vessels shows itself in hæmorrhage, which may also be wide-spread. Yet there are parts

of the body which are favourite seats of hæmorrhage, *e. g.* the brain; indeed, spontaneous cerebral hæmorrhage in middle life, if not due to syphilis, is most frequently found associated with granular kidney. Epistaxis is frequent, and so are small hæmorrhages into the retina. Hæmaturia is not uncommon; it is sometimes copious and long continued. Indeed, I do not think sufficient importance is attached to the continued presence of blood in the urine after what has appeared to be acute nephritis. For my own part I think it a sign of some importance, as indicating the pre-existence of granular kidney.

Of hæmoptysis in granular kidney I know nothing; at least, I have seen no conclusive case. I believe it occurs, and it may be placed in relation with that form of hæmoptysis which the late Sir Andrew Clark described as occurring in gouty persons of advanced age.

Hæmorrhages into the skin are very unusual, and the first of these cases is therefore of great interest. This patient developed, it will be remembered, a condition of purpura, which lasted only for a short time. I have never seen any other case of this kind. She also suffered with hæmorrhage in another most extraordinary place, viz. behind the eyeball. The sudden attack of severe orbital pain attended by some protrusion of the eyeball was in itself suggestive of hæmorrhage, and any doubt that might have been felt at first was set at rest in a few hours by the appearances of subconjunctival ecchymosis. This occurrence, again, is quite peculiar so far as my experience goes.

I have reserved to the last the most remarkable peculiarity of these cases, and that which led to this communication, viz. the *detachment of the retina.* It might have been expected that the detachment was under the circumstances due to hæmorrhage; but, on the contrary, the colour of the affected parts was always pale, and the effusion was clearly serous. In both cases it commenced in the southern or lower segment of the eye, but rapidly extended until it involved the whole periphery except the top or upper segment. Both patients being blind at the time, the detachment manifested itself by no fresh symptom, and was discovered only on ophthalmoscopic examination. It is further to be noted that in the second case the detachment occurred in one eye only, viz. the right, but in the first case it affected both eyes equally.

Detachment of the retina in granular kidney is undoubtedly a very rare affection. How rare it is quite impossible to

say, but my own experience would place it among quite the rarest phenomena, for I have never seen it before out of a very considerable number of cases of granular kidney; and I may add that, knowing the latency of the eye changes in the disease, it has been my routine practice for years to examine the eyes carefully at short intervals in every case of granular kidney which comes under my observation, so that I do not think I am likely to have overlooked instances of it.

XVIII.—*A Novel Form of Malingering: chronic self-inflicted ulceration of the throat.* By Felix Semon, M.D. *Read January* 25, 1895.

ON May 11, 1893, I was consulted by Miss A. C., æt. 36, a weakly and prematurely old-looking lady, on account of "ulcerated throat." The patient's report of her illness, which was corroborated by her mother who accompanied her, was rather remarkable. She stated that she had been suffering for four years or more from almost constant ulcerated sore throat, without any difficulty in breathing and swallowing, and without any change in the voice. Her medical advisers, of whom during the course of her illness she had had many, appeared, according to the description of the patient and her mother, to have all thought at first that they could cure her, but after some time had all declared that they had never met with a similar case, and could do nothing for the patient. One of them was stated to have treated her four years ago with mercurial inunctions to such an extent that she lost all her teeth.

The condition of the soft palate, and to a minor degree of the posterior part of the oro-pharynx, was such as I have never met with before or since. The mucous membrane of the soft palate, the uvula, the arches of the palate, the tonsils, and the posterior wall of the pharynx were one mass of in part apparently submucous whitish infiltration, in part ulceration. In other parts the mucous membrane was bright red, again in others denuded of epithelium; still in others whitish shreds were seen, which in some patches looked like diphtheritic false membranes, in others like the pricked and collapsed skins of blisters; and again in others were yellowish and even brownish in colour, as if strong nitric acid had been applied to them. From the confusing picture no definite idea could be gained as to the real nature of the disease, although of course the possibilities of syphilis and pemphigus suggested themseves; diphtheria, even in its chronic form, being excluded by the enormously long duration of the affection. But the most remarkable thing of all was *the abrupt termination of the affection above and below the region indicated.* Neither in the naso-pharyngeal nor in the œsophageal cavity, nor in the larynx could anything pathological be detected. The nose and the eyes were quite free, and on the skin

no manifestations of disease were found, although the patient stated that sometimes immediately after her bath a red rash appeared over her whole body.

I left the diagnosis in suspense, gave the patient iodide of potassium internally, forbade local applications to the throat of any kind, and requested her to see me again in a fortnight's time.

When seen on May 25 the patient stated that her throat was much better. On examination, however, the condition was in its general aspect similar to that last seen, although *the details* had considerably altered. Some of the spots on the soft palate had completely healed, there were some new rather deep ulcerations with ragged edges on the posterior wall of the pharynx, and some shreds had disappeared from spots in which I distinctly remembered they had been present, whilst fresh ones had made their appearance in parts in which on the previous occasion merely congestion and erosions had been observed. *The most characteristic point about the whole affection, however, remained—its abrupt termination, with almost parallel borders both above and below where the parts would cease to be accessible to the patient herself.*

On my observing that the details of the appearances had much changed, the mother spontaneously stated that such changes frequently occurred from twelve hours to twelve hours.

This unsolicited information finally changed into conviction the suspicion which I had entertained from the very first, viz. that the whole affection was self-inflicted. I sent the patient out of the room, and told the mother that I felt sure that the patient had throughout artificially produced the ulcerations herself by means of nitrate of silver, or some other nitrate compound. This statement, which I was afraid would be received with much indignation, met with a very different reception. The mother was not at all surprised, and on the contrary told me that years ago the patient had artificially blistered her breast, and also at one time blackened her face under her eyes in order to induce sympathy. Mrs. C. also now stated that Mr. Jonathan Hutchinson, who had been amongst the gentlemen consulted, had become convinced that the ulceration of the throat had been artificially produced.

The patient having been called in again, I told her point-blank that I was convinced that the throat affection had been self-inflicted. She denied the fact, but certainly not with the

surprise and indignation a person unjustly accused would show. I then offered to take her for one week's time into a home, where she would be under constant supervision day and night, and that if at the end of that time the ulceration still existed, I was ready to make her my fullest apology for the accusation. She refused the trial under the pretence that her brother was expected home from India, but the mother promised me that after his departure she would do her best to get the girl to consent to it.

Needless to say, I heard no more, and as I was much interested in the case, wrote at the end of October of last year to the mother, and inquired what had become of the affection. From the reply, dated November 2, I take the following sentences:

"I wish I could tell you that the mystery about my daughter's throat was solved. She has seen Dr. K. (the usual adviser of the family) once since she consulted you, but I was not with her, and I am quite sure she did not tell him your opinion. The throat is much the same, and varies from day to day. In July the throat and lips became suddenly very white, and fearing diphtheria, I sent for Dr. K. He assured me it had been caused by some application, probably nitrate of silver, and did not think it necessary to see her again. The whiteness soon passed off."

This is the last I have heard of the patient. Mr. Hutchinson, with whom I had the opportunity of discussing the case on a subsequent occasion, informed me that he too had, on the second occasion when he saw her, become quite convinced that it was a case of malingering. Protean as the morbid power of invention of such patients is to discover new forms of suffering for themselves in order to attract sympathy, and to baffle the skill of their medical advisers, I am not aware that ever the throat has been chosen as the locality for self-inflicted injuries for that purpose, and I therefore consider it my duty to place this case on record.

XIX.—*Cases illustrating the Association of Respiratory Paralysis with Cardio-pulmonary Symptoms in diphtheritic multiple paralysis.* By WILLIAM PASTEUR, M.D.* *Read January* 25, 1895.

I PROPOSE in this paper to dwell more especially on the nature of this relationship in the light of the clinical and post-mortem evidence afforded by 32 cases which have come under my notice since the beginning of 1886. During this period 64 cases (approximately) of diphtheritic paralysis have been treated in the wards of the North-Eastern Hospital for Children, Hackney. In 20 of these, serious cardio-pulmonary symptoms or "crises" occurred, and were associated in no less than 17 of them with paralysis of the diaphragm.

To these 20 cases I have added, for the purposes of the present inquiry, every other case in which there were unequivocal signs of paralysis of the diaphragm or other muscles of respiration, making 32 in all.

With one single exception† this series includes every fatal case of this disease treated in the hospital during the period under review. I may add that eight other cases have been excluded which presented only an incomplete and very transient paresis of the diaphragm.

In a paper published in 1890‡ I drew attention to the remarkable effects on the lungs of paralysis of the chest-walls or diaphragm, and suggested that the lung condition so induced might in some cases be a factor of importance in the production of asphyxial symptoms.

In the following year Dr. Leonard Guthrie§ communicated an interesting paper to the *Lancet,* in which he also referred to the occurrence of diaphragmatic paralysis in what he has aptly named the "bulbar crises of diphtheritic paralysis."

Clinically there is a very striking family resemblance between severe cases of diphtheritic multiple paralysis.

The patients usually present few if any signs of distress

* My best thanks are due to my colleagues at the North-Eastern Children's Hospital, and to Dr. Sidney Coupland, for their kindness in allowing me to examine patients under their care, and to make use of their case-notes.

† This case was left out of consideration on account of the fragmentary and unreliable nature of the notes.

‡ *International Journal of the Medical Sciences,* September, 1890.

§ *Lancet,* vol. i, 1891.

as they lie in bed, for the most part on their back. The breathing is quiet and sometimes rather slow; it may have a sighing character, but is rarely laboured. The face is pale, at times a trifle dusky, and one is forcibly struck by the listless or apathetic condition of the patients. They instinctively shun any kind of exertion. They are with difficulty induced to speak, and then only in a hoarse whisper. Some of them will only nod in answer to questions.

I do not remember ever to have heard one cry. They are generally fairly well nourished, and take food readily, but the swallowing of liquids is often the cause of a good deal of spluttering and regurgitation through the nose, owing to paralysis of the palatal muscles.

Laryngeal paralysis is generally present, and is mainly responsible for the huskiness or aphonia, and in part for the non-explosive nature of the cough from which most of these children suffer. There is *always* more or less paralysis of the extremities, and may be of the trunk and neck muscles. The knee-jerks are always absent. Paralysis of accommodation and of the ocular muscles occurs in some of the cases. On the other hand, paralysis of the palate with characteristic alteration of voice is always present. Weak action of the diaphragm is also not uncommon. Evidence of this is to be found in the extreme feebleness or cessation of respiratory movements at the epigastrium, and in the non-explosive and ineffectual character of the cough.

Some patients who present all these symptoms may, after a variable period, make a complete and uneventful recovery, but in my experience they are specially liable to grave cardio-pulmonary disturbance, of which the following case is a good example.

Alice T., æt. 3 years 10 months, admitted October 17. Diphtheria six weeks ago. "Voix nasillarde" one week. Difficulty in walking four days. Regurgitation of liquids on attempting to swallow.

State on admission.—Fairly nourished. Sallow and rather pale. Tongue furred. Lies quite quiet in bed. Some incoordination of arm movement. Cannot stand without support. Marked ataxia of legs. Pupils equal, no ocular paralysis. Knee-jerks absent; respiration natural. Diaphragm acting normally.

October 24.—Not so well; pulse regular.

October 25.—Immediately after tube-feeding she vomited

and became much cyanosed. Vomiting recurred several times. There is very little movement at the epigastrium. Respiration is chiefly thoracic. Nutrient enemas ordered.

October 26.—Tube-feeding again attempted, but abandoned on account of the sickness it induced.

October 28.—Tube tolerated to-day.

October 31.—Very quiet. Breathing mainly thoracic. Diaphragm still contracting feebly; heart regular.

November 2.—Became very restless an hour after food. Vomited and became cyanosed. Attack passed off during the day.

November 3.—Restless night. Pale and rather dusky this morning. Pulse feeble; respiration entirely thoracic. Epigastrium "much drawn in" during inspiration. At midnight patient suddenly became blue and cold. There was urgent dyspnœa with inspiratory recession at the epigastrium, and increased movement of the lower ribs on both sides. Condition and colour improved after artificial respiration (continued for ten minutes). At 6 A.M. the child was again seized in the same way; she rallied momentarily after brandy and artificial respiration, but sank within two hours. The post-mortem appearances will be dealt with subsequently.

It will be noted that clinically the symptoms, whatever name we may give them, are in the main those of asphyxia, and it will be found that in fatal cases the ordinary signs of asphyxia are usually present.

It is essentially in "bad" cases of multiple paralysis that these accidents are to be apprehended, so that a very general extension of paralysis, and more particularly extreme listlessness and apathy and tendency to sighing respiration, are ominous symptoms.

The analysis of these 32 cases yields the following results:

1. *Age.*—Under one year, 1 case (aged nine months); one to two years, 1 case; two to three years, 4 cases; three to four years, 6 cases; four to five years, 9 cases; five to six years, 7 cases; six to seven years, 2 cases; eight to nine years, 2 cases. Twenty-six out of 32 cases occurred between the ages of two and six years.

2. *History of previous attack of diphtheria.*—In one instance only was there no evidence whatever of a previous illness. In 5 cases the nature of the previous illness was rather indefinite. In the 26 remaining cases the evidence of a previous attack of diphtheria was quite adequate.

3. *Period of paralysis at which cardio-pulmonary symptoms*

supervened.—First week, 8 cases, all fatal; second week, 13 cases, seven died; third week, 5 cases, three died; fourth week, 5 cases, two died; fifth week, 5 cases, three fatal; doubtful, 1 case, fatal.

4. *Vomiting.*—This occurred in 9 of the 19 fatal cases, and in 4 others. In six instances it immediately followed tube-feeding or medicine; in the others no determining cause was apparent.

This symptom was almost invariably associated with an attack of sudden dyspnœa or crisis. Dr. Leonard Guthrie observed it in every one of his cases, and lays some stress upon it. In my opinion vomiting is of bad augury.

Paralysis of diaphragm.—This occurred in 28 cases, of which fifteen ended in death, twelve recovered, and one was lost sight of. In the four remaining cases, which all ended fatally, the cause of death was primarily failure of the circulation.

The duration of the paralysis was, as a rule, much shorter in the fatal cases than in those which recovered.

In four of these cases (Nos. 4, 6, 14, and 16) the intercostal muscles were affected as well as the diaphragm. These cases were of special interest on account of the remarkable lung signs observed, which simulated organic disease (consolidation) of these organs.* In two of them the right intercostals were alone affected, in one the left only, and in the fourth the lower ribs were involved on both sides. It is noteworthy that these four patients all recovered in spite of the extent of the respiratory paralysis. This is probably because they were all instances of peripheral affection of the phrenic and intercostal nerves, whereas the grouping of symptoms in most of the fatal cases is strongly suggestive of affection of the phrenic nerves at or close to their nuclear origin (bulbar crisis).

The *degree* of diaphragmatic paralysis varied considerably, but in every case at one time or other definite reversal of the respiratory movements at the epigastrium was observed. In some instances this was preceded for a day or two by costal breathing and cessation of movement at the epigastrium.

It will have been noted that with three exceptions (Nos. 19, 28, and 29) the diaphragm became paralysed in every fatal case.

6. *Increased movement of lower ribs.*—In fourteen cases

* An altogether parallel case was communicated to the Clinical Society of London in 1876 by Dr. Pearson Irvine, under the suggestive title of "A Case of Diphtheritic Paralysis simulating Extensive Lung Disease."

this phenomenon accompanied the onset of diaphragmatic paralysis.* It is a confirmatory sign of some value, and from its early occurrence may afford timely warning of impending danger.

7. *Nature of the "crises."*—These are for the most part alarmingly sudden in onset. The dominant symptoms are those of asphyxia from failure of respiration and circulation. There is urgent dyspnœa with cyanosis or dusky pallor, and extreme restlessness. The surface and extremities become cold and clammy, or bedewed with sweat. Respiration is laboured, inspiration short and forcible, or gasping, or sighing, and expiration is often feeble. The signs of diaphragmatic paralysis are usually well marked. Mucus rapidly accumulates in the air-passages, and the lungs quickly become œdematous. Rhonchi and râles of varying size are usually to be heard, especially over the bases. In some cases impaired percussion resonance and weak tubular breathing suggest the presence of extensive collapse of the lung. A crisis is often attended by a transient rise of temperature, which may range from one to three, or even four degrees Fahrenheit.

Vomiting occurs in some cases, and may be a distressing symptom.

The paralytic symptoms are, as a rule, distinctly aggravated during the paroxysm.

The condition of the heart and pulse is variable. The pulse-rate is almost always raised to 120, often much higher, and the arterial tension lowered. In some cases the pulse at the wrist is thready and barely perceptible. Irregularity is a common but not a constant feature. It may precede a crisis and disappear with its onset, or become aggravated during the paroxysm. In one or two instances the pulse-rate rose perceptibly on the occurrence of respiratory paralysis, but was not appreciably modified during a subsequent crisis.

Duration and severity.—A crisis may last a few minutes, or be prolonged over two or three days with periods of remission. It may consist of a transient attack of dyspnœa with duskiness of face and extremities, or take the form of an acute suffocative dyspnœa with profound general collapse, and a rapidly fatal issue. Between these extremes every variety and combination may be met with.

8. *Relation of respiratory paralysis to asphyxial crises.*—Paralysis of the diaphragm is met with in these cases under

* The diagnosis of paralysis of the diaphragm in these cases and the value of this sign are fully discussed elsewhere, loc. cit.

two different forms, which are determined, I believe, by the implication of the nuclei and the periphery of the phrenic nerves respectively. It may occur (1) acutely, as part of a bulbar crisis, or (2) in a more insidious form, without other obvious symptoms at the time of onset.

There were seventeen cases of this latter type, in seven of which "crises" subsequently occurred. In the remaining ten cases the paralysis of the diaphragm passed off after a variable interval without further complications.

In the paper already alluded to I suggested in relation to them that the loss of function in the area of lung in contact with a paralysed chest-wall or diaphragm, by embarrassing the pulmonary circulation, as well as through its direct effect on oxygenation, might supply a sufficient cause of asphyxial symptoms, even apart from any direct effect on the heart.

With the evidence now available it seems doubtful whether extensive pulmonary collapse *per se* can be held capable of determining acute asphyxial symptoms such as occur during a well-marked crisis. It is evident from what has been said in reference to the four cases simulating lung disease that very extensive and complete pulmonary collapse is no bar to ultimate recovery. On the other hand, it is found *post mortem* that the extent and degree of collapse appear to vary directly with the completeness and duration of the paralysis of the diaphragm. This, however, is admittedly not an argument in favour of the view that the lung condition may predispose to or determine a "crisis." That it does seriously hamper the heart's action, and may help to determine an attack of heart failure, is highly probable, but with regard to the acute crises I incline to the view that they are probably of central origin.

9. *Mortality.*—Of the 32 cases in the series, 19 died, giving a percentage mortality of 59·3, whilst the mortality for the whole number of cases under treatment (64) during the period under review is 29·6 per cent. The mortality for the 17 cases in which the onset of diaphragmatic paralysis was insidious is 41·1 per cent. (7 out of 17). The mortality among the cases in which crises occurred was very high indeed, 75·4 per cent. (17 out of 22). Of the 5 cases that recovered, the crises were severe in 2, slight in the other 3.

A mortality of close on 30 per cent., even for hospital cases, is probably considerably above the average, and it should be borne in mind that the hospital in which these observations were made is situated in the centre of a district

where diphtheria of a severe type has been prevalent for several years. Still, making due allowance for this fact, and for the limited number of cases here available for statistical purposes, the figures would seem to show that current medical opinion understates the fatality of this disease.

Post-mortem appearances.—An autopsy was performed in eight cases. The organs of circulation exhibited for the most part the usual signs of asphyxia, *i. e.* more or less distension of the right heart with dark fluid blood and black clot, and fulness of the large veins. The lungs require a somewhat more detailed consideration.

Case 3.—Henry H. The diaphragm had been paralysed for six or seven days, and there had been two severe crises, the second of which proved fatal, with signs of asphyxia and heart failure combined.

Autopsy.—Right lung: lower lobe of a deep blue colour, entirely devoid of air. The lung does not crepitate, and is soft and unduly wet. On section a few small patches of broncho-pneumonia are exposed. There is recent pneumonia towards the back of the right upper lobe. The remainder of the right lung is crepitant and œdematous. Left lung: the lower lobe is affected like the right, though in a less degree; the apex is œdematous. Other organs healthy.

Case 8.—William M. was admitted with paralysis of the diaphragm, and died four days later from gradually increasing asphyxia, without having had any definite crisis.

Autopsy.—There were patchy areas of collapse over both lungs with general dilatation of the bronchi, which exuded pus on pressure. There was some interstitial emphysema.

Case 12.—Alfred E. was admitted with marked paralysis of diaphragm, and died on the seventh day with signs of general bronchitis and asphyxia, without definite crisis.

Autopsy.—Right lung: marked collapse of the posterior portion of the lung, more especially of the lower lobe, which is affected almost in its entirety. Pieces of the collapsed lung sink in water. Left lung: this is affected in a precisely similar manner.

Case 18.—Alice T. Described above.

Autopsy.—Right base deep purple and quite airless, sinks entire in water. Bronchi quite clear. Numerous scattered

areas of collapse in left lung, some of considerable size. Some broncho-pneumonia.

Case 20.—Marie V. The diaphragm became gradually paralysed about ten days before death, which occurred after a severe crisis of three hours' duration.

Autopsy.—Both lungs very œdematous. Lower lobe of right lung of a deep purple colour, tough and airless; sinks in water. Lower lobe of left lung partly collapsed. Emphysema of both upper lobes.

Case 27.—Annie E. The diaphragm became gradually paralysed four days before death, which took place after a severe crisis of three hours' duration.

Autopsy.—Right lung: complete collapse of upper lobe with the exception of a small area at the extreme apex. Middle lobe natural. Lower lobe entirely collapsed, and of a uniform deep purple colour. Left lower lobe extensively but not completely collapsed; some crepitation can be felt on pressure.

Case 28.—Edward B. was admitted during a crisis, from which he did not rally. There was no respiratory paralysis.

Autopsy.—There were marked evidences of asphyxia in the heart and large veins. The lungs were œdematous and engorged with dark blood. There was no collapse. The spleen, liver, and kidneys were deeply congested.

Case 31.—David E. The diaphragm was paralysed a few hours before the occurrence of the first crisis. The signs were unequivocal throughout the crisis, but the muscle recovered some power on the following day, and the paralysis did not recur during the final crisis, which was characterised by cyanosis and rapid heart failure.

Autopsy.—Emphysema of upper lobes, and small patches of collapse on the backs of both lungs. Condition of heart not noted.

Before summarising these results the case of William M., whose death was mainly due to purulent bronchitis, may be put on one side. Clinically, too, this case differed somewhat from the paralytic cases.

Seven cases remain for consideration. In one of these (No. 28) no evidence of respiratory paralysis could be found

during life, and in another (No. 31) the paralysis of the diaphragm, though unequivocal, was of very short duration. In the first of these two cases there was no trace of pulmonary collapse, and in the second only a few small patches along the posterior borders.

In the five remaining cases there had been paralysis of the diaphragm of some duration (two to four days at least), and in every one of these the lungs were profoundly affected.

In four the collapse of the right base was "complete;" in the fifth it was "almost complete." In every case the right lung was affected more than the left. There were no pleural adhesions, pleural effusion, or obstruction of the trachea or bronchi in any of the cases.

It cannot be doubted, I think, that the state of the lungs is the direct result of the paralysis. The varying relation to the degree and duration of paralysis points strongly to this conclusion, which is further strengthened by the signs of consolidation of lung observed in the cases simulating lung disease.

Further, the fact that the lung signs always occur in that portion of the organs which is in immediate relation to the paralysed chest-wall, and apparently in no other, makes it difficult, if not impossible, to accept the view that they are in any measure due to paralysis of the bronchial muscles.

But although immobility of the chest-walls is well known to be an efficient cause of collapse of lung, the wholesale way in which this occurs in these cases has always struck me as being very remarkable. I know of no condition—unless it be a very large pleural effusion—which will empty a lung of air as completely as this.

The greater vulnerability of the right base is somewhat perplexing; it may possibly be due to the different anatomical relations of the pulmonary bases to the diaphragm and subjacent viscera.

In conclusion the more salient points touched upon may be summarised as follows:

1. That the mortality in diphtheritic multiple paralysis is probably higher than current opinion would lead one to suppose.
2. That death takes place by asphyxia.
3. That the fatal symptoms are in the large majority of cases of sudden onset, and suggest a central origin.
4. That recovery from a "bulbar crisis" is exceptional.
5. That paralysis of the diaphragm is comparatively fre-

quent, and may occur either acutely during a bulbar crisis, or may develop insidiously as part of the peripheral paralysis.

6. That the supervention of respiratory paralysis very seriously increases the gravity of the prognosis.

7. That whenever paralysis of the diaphragm or other part of the chest-wall is long continued (two or more days), collapse of the adjacent portion of lung is very liable to occur, and may give rise to definite physical signs.

8. That the base of the right lung appears more liable to be affected in this way than the left.

I append below a very brief summary of the cases on which the foregoing analysis is based.

Case 1.—H. D., æt. 5 (F.), admitted April 19, 1889. Died. Diphtheria two months ago. Nasal twang one month. Weakness of legs three weeks. Regurgitation of fluids through nose three days, with loss of power to cough.

April 19.—Dull and drowsy. No dyspnœa. Voice weak and nasal.

April 20.—Diaphragm paralysed. Numerous moist râles over lungs. Heart weak, irregular, intermittent; pulse 140. Vomited three times. Paralysis of palate and right external rectus oculi.

April 21.—Pulse better. Diaphragm still paralysed; lungs full of mucus. Unable to swallow. At 1 P.M. became very restless and fainted; recovered on being inverted. Vomited three times.

April 22.—Voice stronger; sick four times. Very quiet to-day. At 8 P.M. became suddenly restless and livid; condition did not yield to treatment, and death occurred at the end of a few hours. No post-mortem.

Case 2.—H. B., æt. 3 (M.), admitted June 4, 1888, removed by parents. No history of previous diphtheria. Ineffectual cough fourteen days. Nasal twang noticed one week.

June 4.—Characteristic, weak, ineffectual cough. Breathing almost entirely thoracic, hardly any movement at epigastrium. No dyspnœa; sonorous and sibilant rhonchi over chest; pulse 120. Heart acting weakly. Palate paralysed. Knee-jerks absent. Swallows without difficulty.

June 6.—Decidedly weaker; the diaphragm is completely paralysed.

Note.—His mother states that after removal from hospital the child slowly recovered at the end of two months. During this period he remained very pale, and his cough was noiseless, but there was never any difficulty in swallowing.

Case 3.—H. H., æt. 4 (M.), admitted August 28, 1888. Died. Acute throat affection six weeks ago with discharge of "blood and matter" from mouth. Acute abscess of tonsil one month ago, followed by stomatitis. Nasal twang noticed on August 17. Weakness of limbs since August 20.

August 28.—Marked anæmia and great general weakness. Heart irregular and intermittent. Pulse 120; respiration quiet, 16 per minute. Knee-jerks absent, superficial reflexes present. Urine removed by catheter, normal.

August 30.—Heart irregular and tumultuous last night. Breathing deep, rather slow, entirely thoracic; epigastric recession during inspiration, no movement of viscera discernible; paralysis of diaphragm apparently complete. Heart rapid, regular, sounds sharp.

August 31.—Very restless night; laboured breathing with some cyanosis. Pulse very small, too rapid to count. Urine contains much albumen and urates; no casts. *Later.*—Pulse 204; air enters well into upper part of lungs, but feebly at bases and along anterior margins. No râles.

September 3.—Looks better; pulse has fallen to 138, fair tension. Breathing still costal; resp. 36. Epigastrium still falls in during inspiration. Paralysis of limbs very marked. Is unable to cough. Loss of control over bladder. Temperature below 100°.

September 4.—Temperature last evening 100·6°; at 4 P.M. patient suddenly became very pale and anxious, respiration became rapid and laboured, and the pulse irregular (144). At the same time the epigastric recession became more marked than hitherto. Condition improved after a few hours, but heart continues irregular.

September 5.—Temperature rose to 103° this evening, and signs of consolidation at right base were made out (fine crepitations). Very restless; pulse 168, resp. 60.

September 6.—Increasing dyspnœa and loss of strength since yesterday. Death by asphyxia.

Post-mortem examination.—Heart uncovered. Right lung: lower lobe of deep blue colour, entirely devoid of air. No adhesions. Does not crepitate on section. Numerous patches of broncho-pneumonia scattered through the collapsed lung tissue. Lung unduly soft and wet. Recent pneumonia of posterior portion of upper lobe, remainder of right lung crepitant and œdematous.

Left lung: lower lobe affected in same manner as right, though in a less extreme degree. Upper lobe œdematous. Heart muscle firm and pale. Other organs look healthy.

CASE 4.—W. B., æt. 4½ (M.), admitted September 11, 1888. Recovered. Diphtheria six weeks ago. Regurgitation through nose three weeks. Weakness of legs four days.

September 12.—Pale, quiet, general muscular weakness; ataxic tottering gait. Paralysis of palate, nasal voice; knee-jerks absent, superficial reflexes present; urine normal; respiration normal, diaphragm acting well; lungs healthy.

September 14.—Pulse 120, some irregularity, resp. 18, sighing in character; slight difficulty in swallowing, followed by cough. Some increase in movement of lower ribs, and some diminution of the normal movements at epigastrium.

September 17.—Slight impairment of resonance over both bases with scanty mucous râles. Pulse as above.

September 24.—Excursion of lower ribs much increased. No movements of abdominal viscera at epigastrium; diaphragm seems to be quite inactive. Temperature about 99°; definite signs of consolidation (? collapse) of right base without râles, except on deep breathing. Frequent sickness, especially after brandy.

September 25.—No movement at epigastrium. Tympanitic note as high as sixth rib in right nipple line. Lung note down to fourth rib; breathing very weak; cough non-explosive.

September 28.—Condition the same; profoundly anæmic. Breathing

essentially upper costal; some recovery of power in cough; no more regurgitation; physical signs still present at right base; temperature about 99°.

October 1.—Distinct respiratory movements at epigastrium, which coincide with a general amelioration of symptoms. Signs much less at right base, where air now enters well. Convalescence was uninterrupted from this time.

Treatment.—Artificial respiration several times a day.

Case 5.—A. B., æt. 4 (M.), admitted December 18, 1888. Died. Sore throat five weeks ago; in bed a fortnight; weakness of limbs five days. Nasal voice two days. Weakness increased steadily during first five days in hospital without salient symptoms.

December 17.—Extreme weakness; diaphragm acting feebly, but not paralysed; cough explosive; temperature subnormal; pulse 144, regular, rather feeble. Some mucous râles over both backs.

December 18.—Slight percussion dulness over left base; diaphragm still acting, though feebly. Towards evening patient turned very blue. On examination the diaphragm was found to be paralysed, with reversal of respiratory movements at epigastrium. Pulse weaker and more frequent, temperature 100°; signs of consolidation in left axillary region. Child died a few hours later, temperature falling before death to 97°. No post-mortem.

Case 6.—J. H., æt. 6 (M.), admitted March 11, 1889. Recovered. Diphtheria five weeks ago. Tottering gait and wasting one week. Regurgitation through nose three days.

March 11.—Anæmic, cannot stand alone; knee-jerks absent; superficial reflexes present. Lower ribs act vigorously, and abdominal wall falls in during inspiration. Voice somewhat nasal, fairly strong cough. Some difficulty in swallowing. A few large moist râles over left back; pulse 120, regular; temperature up to 90°.

March 15.—Last evening pulse became irregular and intermittent.

March 16.—Sick directly after tube-feeding, and began to gasp for breath and became blue with failing pulse. Recovered after two or three hours, during which stimulants and artificial respiration were freely resorted to. Temperature rose to 99·6°.

March 17.—Temperature normal. No sickness. Moist râles over both bases.

March 18.—Voice and cough weaker. Movement of right half of chest just appreciable, breathing barely audible at right base.

March 19.—Paresis of both external recti.

March 20.—Right internal rectus affected. A little sick this morning. Patch of bronchial breathing in right axilla, with faint bronchial breathing over remainder of right base. Evening temperature yesterday 100°, to-day 99·4°.

March 22.—Decided improvement in general condition. Tube-feeding omitted. Well-marked signs of consolidation (dulness and bronchial breathing) at left base. Breath-sounds somewhat bronchial at left apex below clavicle. Temperature remains about normal.

March 23.—Condition of bases unchanged.

March 24.—Swallows perfectly. Movement of right side of chest still very limited.

March 26.—Some recovery of power in soft palate.

March 29.—Some movement of epigastrium noticed to-day at commencement of inspiration. Cough is again slightly explosive.

April 2.—Diaphragm now contracting feebly.

April 5.—Diaphragm contracting well.

April 8.—Breathing still weak at bases, but all signs of consolidation have disappeared.

April 16.—To get up. Faradic irritability of leg muscles notably diminished. No R. D.

Note.—Artificial respiration was practised thrice daily for ten or fifteen minutes until March 28.

Case 7.—A. M., æt. 3 (F.), admitted May 14, 1889. Died. Admitted on account of chronic eczema of head and limbs. Feverish and ill fourteen days ago with enlarged glands below jaw. Some feebleness of limbs since two days. No additional evidence of paralysis until June 6, when patient was unable to stand. More fretful since three days. Knee-jerks absent, superficial reflexes present.

June 11.—Double ptosis, sluggish unequal pupils. No affection of diaphragm or intercostals; palate paralysed; loss of power in cough.

June 14.—More irritable. Condition not unlike meningitis. Some cardiac irregularity yesterday. A few moist sounds at right base.

June 17.—Movement of lower ribs rather exaggerated. Rhonchi and moist râles over both bases. Sick twice after medicine.

June 20.—Sick this morning. Swallowing less satisfactory than yesterday and followed by cough. More drowsy. There is inspiratory recession at epigastrium, with definite over-action of lower ribs. Heart very irregular. Râles over bases more abundant. Cough and voice very feeble. Patient grew worse throughout the day with increasing cyanosis, and died the same night. No post-mortem. The temperature remained low throughout.

Case 8.—W. E. M., æt. 2½ (F.), admitted June 19, 1889. Died. Indefinite illness one month ago. Regurgitation of food through nose one week.

June 19.—Anæmic. No distress. Right internal squint. Knee-jerks absent, superficial reflexes present. Nasal voice; much cough after swallowing. Increased action of lower ribs and epigastric recession with inspiration. Moist râles all over lungs; no dulness. Heart-sounds weak. Temp. 98·2°.

June 22.—Temperature rose last evening to 103°. No fresh physical signs to-day, but is much weaker in every way. Died towards evening.

Autopsy.—A few small areas of collapse at bases of both lungs. General moderate cylindrical dilatation of bronchi with exudation of muco-pus on pressure. Some interstitial emphysema. Other organs apparently healthy.

Case 9.—B. R., æt. 5 (F.), admitted June 22, 1889. Removed. Nasal discharge one month ago with febrile symptoms. Nasal voice fourteen days. Inability to walk one week.

June 22.—No distress; frequent non-explosive feeble cough. Double ptosis. Paresis of both external recti. Distinct over-action of lower ribs, and evident paralysis of diaphragm. Air enters bases well. No dulness or râles. Heart normal. Urine lithatic: trace of albumen.

June 25.—Ptosis more accentuated. Diaphragm still paralysed. Temperature subnormal.

Note.—Patient was removed by the mother next day, and her subsequent history could not be traced.

Case 10.—P. S., æt. 2 (M.), admitted August 12, 1889. Recovered. Diphtheria five weeks ago. Inability to walk since ten days. Nasal voice three days.

August 12.—No distress, but fretful. Drinking excites cough. Palate motionless. Cough wanting in strength. Knee-jerks absent, superficial reflexes feeble. No albuminuria. Râles and rhonchi all over chest. Heart healthy. Temp. 98·6°.

August 14.—Cough troublesome. Fed through nasal tube.

August 16.—Last night, after a paroxysm of coughing, breathing became hurried and the surface rather livid. The diaphragm was acting, but there seemed to be excessive movement of the lower ribs. Temperature rose from normal to 101·2°. Temperature this morning 102·8°. Sick after tube at 7 A.M., and shortly became blue and cold. The diaphragm was seen to be paralysed. After artificial respiration for fifteen minutes the colour was better and the distress was over. The epigastrium remains motionless except for a tendency to fill up a little during expiration. The ribs are moving very freely. Evening temperature 102·6°.

August 19.—Improving. Epigastrium still motionless. No physical signs in lungs.

August 20.—Some signs of returning activity of diaphragm last evening.

August 24.—Abdominal movements of respiration now fairly normal though weak. Cough regaining explosive quality, and speech improving.

August 29.—Cough quite natural. Diaphragm acting normally, movements of lower ribs far less obtrusive. Rapid recovery from this date.

Case 11.—A. G., æt. 8 (M.), admitted August 21, 1889. Recovered. Diphtheria six weeks ago, anæmic since; nasal voice ten days; regurgitation of food through nose three days.

August 21.—Anæmic; palate paralysed; very quiet, and free from distress; temp. 99·2°; heart and lungs healthy; weakness of both external recti; knee-jerks absent, superficial reflexes present.

August 28.—Cough grew weaker yesterday, and is non-explosive to-day; rather restless and irritable; evening temp. 99·6°; abdominal movements very slight, whilst action of lower ribs has become exaggerated; no dyspnœa, fair colour.

August 29.—Cough after swallowing since last night; weak breathing at right base.

August 30.—Slight increase of abdominal respiratory movements.

September 2.—Abdomen moves well to-day; rib movements quieter; cough still non-explosive.

September 18.—Voice nearly normal.

Case 12.—A. E., æt. 2 years 4 months (M.), admitted September 18, 1889. Died. Yellow discharge from nose seven weeks ago, with snuffly voice and regurgitation through nose.

September 18.—Very apathetic and still, face pale and dusky; cough non-explosive; knee-jerks absent; epigastrium and belly wall blown outwards in expiration; diaphragm appears to be quite passive; costal respiration; moist râles all over lungs. Breathing faintly bronchial at left apex; no heart disturbance; urine albuminous. Temp. 101·2°.

September 23.—Numerous moist râles over right back; accumulation of mucus in throat and mouth. Breathing became more embarrassed at 6 A.M.

with increase of lividity; temporary improvement with artificial respiration. Movement of lower ribs exaggerated to-day; asphyxia gradually increased, and death occurred on morning of 24th.

Autopsy.—No pleural adhesions; marked collapse of posterior aspect of right lung, especially of lower lobe, which is involved almost in its entirety. The posterior part of the right upper lobe is also completely collapsed; portions of the collapsed lung sink in water. The left lung is affected in a precisely similar manner; right auricle somewhat distended with clot, valves healthy. Liver and kidneys congested.

Case 13.—W. W., æt. 4 (M.), admitted October 24, 1889. Died. Sore throat five weeks ago. Difficulty in walking one week.

October 24.—Fair colour; rather fretful; cannot stand alone; no paralysis of palate or difficulty in swallowing; knee-jerks absent; plantar reflex present; heart and lungs healthy; no respiratory paralysis.

October 29.—Difficulty in swallowing; palate paralysed.

November 2.—Pulse 96°, fair quality.

November 6.—Slight right internal squint and limited lateral movements of both eyes; voice nasal; very quiet; excessive movement of lower ribs, and marked lessening of epigastric movement in respiration.

November 8.—Cough still retains some power; slight expiratory bulging of epigastrium.

November 9.—Cough weaker; pulse 88.

November 11.—Distinct recession of abdominal wall during inspiration; no albuminuria.

November 14.—Was restless and paler last evening, and breathing became slower (? sighing); some paralysis and inco-ordination of arms; breathing continues entirely thoracic with reversed movements at epigastrium. No physical signs over pulmonary bases; pulse 124, very compressible.

November 15.—Breathing slow and sighing in character, no dyspnœa, eighteen per minute. At 11 A.M. patient vomited without apparent cause, and became dusky, with gasping respiration and very frequent pulse. Rallied somewhat after stimulation. Sick again at 2 P.M.; obvious failure of circulation. Faint bronchial breathing at extreme base (both sides), which soon became obscured by copious moist râles (œdema). 4 P.M., livid, and sweating profusely, marked dyspnœa; extreme movement of lower ribs, and sucking in of epigastrium during inspiration, which flaps outwards in expiration; slight inspiratory recession of lower intercostal spaces. Death at 4.15 p.m. Temp. never above 99°. No post-mortem.

Case 14.*—J. H. was admitted in May, 1886, with well-marked diphtheritic paralysis of the trunk and limbs. On the tenth day some irregularity and increased frequency of pulse were noted. Meanwhile the paralysis was becoming more profound, and the cough was altogether ineffectual and non-explosive. There is no detailed note as to the character of the respiratory movements, except that "the movements of the ribs appeared to grow less day by day."

On the seventeenth day respiration suddenly became embarrassed, and lips and extremities cold and bluish. Examination of the chest showed that the right side was practically immobile, with the exception of the two or three upper ribs. The percussion note was much impaired over the right front and base. At the apex in front the breathing was loud and tubular,

* For details of this case see 'Lancet,' 1887.

weak over the middle lobe, and almost inaudible at the base; behind, above the level of the scapular angle, the breath-sounds were fairly loud and bronchial. There was no pain of any kind, and the temperature was not raised more than a degree. On the eighteenth day the child was much weaker; there were impaired resonance and weak tubular breathing, without râles, all over the right front, and exaggerated breathing over the left lung. The heart was not displaced; the backs were not examined. The movement of the right half of the chest was only just appreciable, and the epigastrium receded during each inspiration, whilst it bulged slightly during expiration, and more distinctly so during cough, which was very frequent and ineffectual. The temperature was beginning to rise (100°), the pulse 144 and more thready. During the next two days the child grew worse, and the temperature rose gradually to 102·4°. On the twenty-first day, however, the case began to look more hopeful. The change for the better was rather sudden, and amongst the first favorable indications were a return of movement in the right chest and a rapid disappearance of the signs of diaphragmatic paralysis. Examination of the backs revealed bronchial or tubular breathing over the right apex and posterior apex of the lower lobe, and distant weak breathing at the extreme base of the right side and exaggerated breathing over the left. On the twenty-fifth day the two sides of the chest moved equally. Three days later the breathing at the right apex had lost its tubular quality.

Case 15.—J. R., æt. 3 (M.), admitted December 9, 1889. Recovered. No history of diphtheria. Weakness of legs one week.

December 9.—Very quiet, refuses to talk. Knee-jerks absent. No paralysis beyond weakness of legs. Heart and lungs healthy. Temp. 100°.

December 25.—Cough wanting in power to-day, and more frequent; often follows swallowing.

December 26.—More fretful, voice very weak. Cough almost non-explosive; no dyspnœa; resp. 20; pulse quiet, 92; temp. normal. Abdominal movements of respiration, which have hitherto been well marked, have almost ceased, and the slight movements observed are reversed in order. Lower ribs are moving normally.

January 2.—Swallows better; still some regurgitation through nose. Abdomen passive.

January 8.—Marked improvement. Normal abdominal movements have reappeared since two or three days. Convalescence uninterrupted from this date.

Case 16.—W. C., æt. 4½ (M.), admitted November 3, 1893. Recovered. Diphtheria five weeks ago. Weakness of legs one week. Nasal voice and regurgitation of food two days.

November 3.—Anæmic. Nasal voice. Palate paralysed. Difficulty in swallowing. Knee-jerks absent. Heart normal. No respiratory paralysis.

November 8.—Troublesome regurgitation.

November 11.—Refusing food. Pulse weak. Cough non-explosive. There is some diaphragmatic breathing, but expansion of left chest is almost *nil*. Manifest depression and flattening of left chest, confirmed by cyrtometer. Higher pitched percussion note than over right side. Breathing almost absent over left, and exaggerated over right lung. Heart's apex-beat in fourth space outside nipple-line. Temperature rising gradually since 9th, now 100·2°.

November 13.—Lips rather dusky. Coarse tracheal or bronchial gurgling to be heard with respiration. Cough abortive. More movement and improved resonance in left chest in front, but left back is duller, with bronchial breathing at scapular angle. Here moist crepitations.

November 14.—Movements of diaphragm very feeble. Some blocking of lungs with mucus. Pulse fair.

November 23.—Steady improvement; temperature normal again to-day.

November 28.—Temp. 101·6°. Measles rash. Removed to an isolation ward. No notes of chest condition until December 29, when all signs of diaphragmatic paralysis had disappeared. Chief movement is in lower third of chest. Left chest moves decidedly less than right. Breathing weak all over left lung, and bronchial over front and axilla. Crepitations in latter situation on deep inspiration. Exaggerated breathing on right side. The chest returned to a normal state towards the end of January. The knee-jerks had returned on March 1st.

Case 17.—N. S., æt. 1½ (M.), admitted June 9, 1892. Died. Sore throat six weeks ago. Loss of power in legs four weeks. Altered voice one week. Regurgitation of fluids four days.

June 9.—Quiet and listless; anæmic; knee-jerks absent; superficial reflexes present; paralysis of palate; epigastrium "drawn inwards" in inspiration. No physical signs in lungs; heart rapid, rather weak.

June 11.—Unable to swallow—tube-feeding.

June 13.—Pulse 168, small; respiratory movements at epigastrium reversed; pupils sluggish; no albuminuria.

June 14.—Alternately flushed and pale; weaker; irritable when disturbed.

June 15.—Marked paralysis of diaphragm. At times cyanosed during tube-feeding.

June 17.—Very pale; respiration feeble and irregular. No action of diaphragm; weak breathing at bases with some moist râles. Became cyanosed during afternoon, with slow gasping respiration; heart continued beating after cessation of breathing. No post-mortem.

Case 18.—A. T., æt. 3½, (F.). (For notes of this case *vide sup.*, p. 112).

Autopsy.—Body well nourished.

Heart.—Right side dilated and full of dark clot, otherwise healthy.

Lungs.—Right: pleura normal; no adhesions. The whole lung, excepting a few groups of vesicles on the front of the middle lobe, is of a deep purple plum colour, does not crepitate, and sinks entire in water. On section no signs of broncho-pneumonia or of bronchial obstruction. Left: scattered areas of collapse, some of considerable size. One or two small patches of broncho-pneumonia. Several enlarged glands in mediastinum, one caseating; some enlarged mesenteric glands. Other organs appear healthy.

Terminations of phrenic nerves in diaphragm soaked for twelve hours in Müller's fluid, then thoroughly washed and teased, show no signs of peripheral neuritis after staining with osmic acid.

Case 19.—H. R., æt. 4½ (M.), admitted January 4, 1892. Died. Ulcerated throat three weeks ago. Weakness of back ten days.

On admission.—Knee-jerks absent, but no other evidence of paralysis. Heart and lungs healthy; pulse 120; voice natural.

January 7.—Pulse 128, slightly intermittent; some difficulty in swallowing.

January 14.—Losing ground the last two or three days; voice nasal; feeble cough often follows swallowing. Rhonchi over lungs; air enters well everywhere.

January 15.—At 11 A.M. became cyanosed, and movements of chest seemed diminished. Pulse 120, regular. Much better after dinner. At 4 P.M., another attack of cyanosis, and death within two minutes. No post-mortem.

CASE 20.—M. V., æt. 4 (F.) admitted March 16, 1893. Died. Indefinite febrile illness since one month. Difficulty in swallowing and nasal voice fourteen days. Weakness of legs four days.

March 16.—Poorly nourished; nasal voice; cough at times after swallowing. No albuminuria. Palate paralysed. Knee-jerks absent. Pupils unequal. Diaphragm not acting fully. Heart and lungs healthy.

March 29.—Condition stationary for first ten days; now has more cough after swallowing. Pupils equal. Weakness of both external recti oculorum. Diaphragm acting feebly. Ankles dropped; knee-jerks absent. External jugular veins dilated. First sound toneless, second accentuated. A few moist râles over lungs.

March 31.—Pulse 140. Drinks with difficulty. A little inspiratory recession at epigastrium. Is generally weaker. At 11 P.M. the nurse found patient livid and gasping. Breathing had ceased when house surgeon arrived. Child rallied after stimulants and artificial respiration, and breathed spontaneously for about ten minutes so long as the tongue was drawn forwards. Life was maintained for two hours by artificial respiration without any return of consciousness. Heart-beats continued for some seconds after cessation of respiration.

Autopsy.—Chest thinly covered; muscles wasted; trachea injected; contains much viscid mucus. A small piece of "membrane" quarter of an inch long in one of the bronchi. Lungs œdematous; right base airless, smooth, and deep purple; sinks in water. Left base partly collapsed. Emphysema of both apices; no pleural adhesions or effusion. Right heart and large veins distended with dark fluid blood; valves healthy. Other organs healthy.

CASE 21.—J. G., æt. 2½ (F.), admitted February 9, 1893. Died. Diphtheria about middle of January. Weakness of legs and sickness after food ten days. Ocular paralysis and loss of accommodation eight days. Nasal voice one week. Unable to stand three days.

February 9.—Very prostrate. Pulse 136, regular, fair. Respirations 24. Laboured and excessive action of lower ribs; reversed movements at epigastrium. Knee-jerks absent. Harsh breathing at apices, feebler towards bases, with copious moist râles.

February 10.—Worse. Voice more nasal. Much restlessness. Bad attack of dyspnœa early this morning, relieved by artificial respiration.

February 11.—More râles—some finer—over bases; no dulness made out. Temperature rose next day to 101° before death, the mode of which is not noted. No post-mortem.

CASE 22.—J. R., æt. 5 (F.), admitted January 31, 1893. Recovered. Diphtheria in middle of December lasting four days; nasal voice three weeks; tottering gait a few days.

January 31.—Fair colour; nasal voice; non-explosive cough; swallowing slow and difficult; internal squint; paralysis of palate; weakness of

legs. Movements of thorax in respiration free, especially below, where they are excessive. Resp. 24, sighing at times. Diaphragm acting very feebly; more strongly in coughing; heart 130, regular; some fulness of external jugular veins; harsh breathing at apices of lungs; weak breathing, with numerous moist râles and rhonchi over bases. No albuminuria. Knee-jerks absent.

February 7.—Speaks and drinks better. Action of diaphragm much stronger, normal though feeble movements at epigastrium. Ocular paralysis less. Lungs as before.

February 11.—Diaphragm acting normally. Rapid convalescence.

Case 23.—H. C., æt. 5 (F.), admitted April 10, 1893. Recovered. Diphtheria at end of February. Nasal voice four weeks, with some regurgitation. Troubles of vision one week.

April 10.—Well nourished; nasal voice; paresis of legs. Difficulty in swallowing. Frequent sickness. Rapid pulse, 144, regular. Very weak action of diaphragm, barely perceptible movement at epigastrium. Knee-jerks absent. Temperature normal throughout.

April 17.—Steady improvement. Voice natural, swallows easily. Less cough after drinking. Pulse 120. Diaphragm acting more forcibly. Moderate inspiratory protrusion of epigastrium. Lungs healthy.

April 24.—Pulse 96. Convalescent.

Case 24.—M. C., æt. 5 (F.), admitted July 24, 1893. Recovered. Sore throat two months ago. Squint one week, with nasal voice and difficulty of swallowing. Tottering gait a few days.

July 24.—Râles and rhonchi all over chest, with dulness at left base. Nasal voice, marked squint, much spluttering on swallowing liquids. Knee-jerks absent. Diaphragm acting very feebly, if at all. Thoracic movements very free. Urine acid, one tenth albumen. Sudden attack of extreme weakness and inability to swallow at 7 P.M. Relieved by brandy and digitalis.

July 26.—Sudden attack of coughing at 11.45, followed by severe spasm of larynx.

July 27.—Laryngeal stridor since yesterday. Diaphragm very feeble. (Tracheotomy instruments kept in readiness.)

July 28.—Severe attack of coughing last night, and again to-day.

July 29.—Diaphragm acting more strongly; still some laryngeal stridor. Recovery rapid from this date. (? broncho-pneumonia and pertussis.)

Case 25.—G. A. S., æt. 3 (M.), admitted November 30, 1893. Died. Diphtheria a month ago. Since one week difficulty in walking and nasal speech.

November 30.—Unable to stand; squint; nasal voice; paralysis of palate; knee-jerks absent; diaphragm active; regurgitation of liquids. Temp. 99°.

December 2.—Very restless. Heart irregular, 124. Respiration not quickened.

December 3.—Tube-feeding—sick after first attempt. Continues in an irritable state. Crepitations and rhonchi over both lungs, especially at right base. No dulness. Resp. 32; pulse 120.

December 4.—Pulse 116, still irregular. Frequent non-explosive cough, much mucus in pharynx.

December 5.—Very exhausted after tube-feeding, pulse 184.

December 7.—At 6 a.m. found to be livid, and breathing badly with rattling in throat. Diaphragm inactive. Inspiratory recession marked at epigastrium. Condition improved temporarily by artificial respiration. Death a few hours later. No post-mortem.

Case 26.—E. M., æt. 9 months (F.), admitted February 12, 1894. Died. Recently discharged from North-Eastern Fever Hospital after diphtheria.

On admission.—Pallid. Ill-nourished. Paresis and wasting of limbs. Knee-jerks absent. Regurgitation after swallowing. Chest expands well. Heart-sounds healthy.

February 14.—Temperature has risen to 101·2°. Unable to swallow. Marked epigastric recession. A few crepitations at bases, no signs of consolidation. Paralysis of diaphragm became still more marked during the day. Died that evening. No post-mortem.

Case 27.—A. E., æt. 5½ (F.), admitted April 30, 1894. Died. Diphtheria a month ago. Nasal voice, regurgitation through nose, and weakness of legs one week.

April 30.—Breathing quiet, cough non-explosive. Very listless. Palate paralysed; swallowing difficult. Knee-jerks absent. Chest expands freely, but action of diaphragm feeble. Heart-sounds clear; pulse regular, fair quality.

May 3.—Getting weaker. Lateral movements of eyes lost; accommodation sluggish. Cough very feeble. Movements at epigastrium reversed. Weak breathing at right base.

May 6.—Paralysis of diaphragm well marked. Movement of lower ribs increased. Breathing very faint at right base. At 2.30 P.M. suddenly became cyanosed, and chest movements were found to be much diminished. Artificial respiration was tried without success. Death at 3.30.

Autopsy.—No pleural adhesions or effusion. Right lung: upper lobe collapsed with exception of a small area at the extreme apex; middle lobe natural; lower lobe entirely collapsed, of a uniform deep purple colour, and unduly wet. Left lung: Upper lobe natural. Lower lobe extensively collapsed, but not entirely. Trachea and bronchi injected, and full of mucus. Pericardium healthy. Right heart distended with dark fluid blood. Left side empty and contracted. Myocardium healthy. Liver, spleen, and kidneys congested.

Case 28.—E. B., æt. 6 (M.), admitted August 3, 1886. Died. Sore throat three weeks ago.

On admission.—Very collapsed. Pale, with dusky lips. Cold extremities. Pulse barely perceptible. Distended jugular veins. Laboured breathing with deep sighs at frequent intervals. Thoracic and abdominal movements normal. No paralysis of diaphragm. Temperature subnormal. No physical signs in lungs. Is able to swallow. Patient recovered somewhat after getting to bed, and took food and stimulants. A few hours later signs of asphyxia reappeared, and death took place the same night.

Autopsy.—Veins much distended with dark fluid blood. Heart distended with blood on both sides; mixed clot in both ventricles. Lungs deeply

congested with dark blood, and very œdematous; no collapse. Liver and kidneys congested. Congested cerebral membranes.

Case 29.—W. W., æt. 4 (M.), admitted November 13. Sore throat and "quinsy," seven weeks. Nasal voice, regurgigation, and altered cough two weeks.

November 13.—Pale, quiet, distressed look. Breathing quiet. Frequent non-explosive cough. Feeble pulse. Knee-jerks absent; unable to stand. Diaphragm normal.

Fed by tube at 7 P.M.; brought on coughing, vomiting, and some lividity. Within three minutes became cyanosed and unconscious. Pulse began to fail two or three minutes later. Condition momentarily improved by artificial respiration and stimulation. Death one hour later. No P.M.

Case 30.—R. B., æt. 8 years (F.), admitted November 18, 1892. Recovered. Diphtheria seven weeks ago. Weakness of legs, with nasal voice and regurgitation three weeks. Impairment of vision fourteen days.

November 18.—Pale, quiet, palate paralysed. Cough non-explosive. Chest movements normal in quiet breathing. Diaghragm paralysed. Reversed movements at epigastrium. A few bronchitic râles in lungs. Knee-jerks absent.

November 21.—Sick yesterday after tube-feeding.

November 25.—Voice stronger. Diaphragm acting slightly. Cough less.

November 28.—Diaphragm recovering steadily.

December 9.—Cough explosive again. General improvement. Able to walk.

Case 31.—D. E., æt. 4 (M.), admitted January 18, 1887. Died. Diphtheria five weeks ago. Increasing weakness since one week.

On admission.—Pale and apathetic. Breathing quiet (22). Pulse regular, 145. Occasional feeble cough. Respiratory movements those of health. Rhonchi over both backs. Palate paralysed, difficulty in swallowing. Large trace of albumen in urine.

January 11.—Cough after swallowing. Pulse very weak, but regular.

January 12.—Pulse 156. Cough non-explosive. Breathing slow and laborious, inspiratory recession at epigastrium. Sick at 3 P.M. after beef-tea, and became blue and faint. Epigastric recession more marked, "but viscera can be felt to descend during inspiration" (?) Pulse 128, irregular.

January 13.—Pulse again regular and diaphragm active. Expansion of lower half of chest good. In the afternoon cough became more frequent, and swallowing more difficult. Control was lost over sphincters, and towards evening the patient became unconscious, with weak pulse of 140. Death occurred at 9 P.M.

Autopsy.—Right lung: emphysema at apex and along anterior border; several small patches of collapse on posterior aspect. Pleuræ healthy. Left lung: slight emphysema in front; numerous small areas of collapse all over posterior aspect and base Heart: some dilatation of left side (*sic*). Valves competent.

Case 32.—E. C. P., æt. 3½ (F.), admitted June 21, 1891. Died. Febrile affection with lumps in neck and nasal discharge seven weeks ago. Weakness of legs three weeks, also cough on swallowing and regurgitation of liquids through nose.

On admission.—Looks moribund. Pale and dusky; eyelids half closed; pupils dilated; tongue protruding; jactitation. Rapid weak pulse; temperature 101°. Frequent non-explosive cough. Rattling in trachea. Rapid shallow breathing. Cold extremities. Rallied considerably after stimulation and warmth.

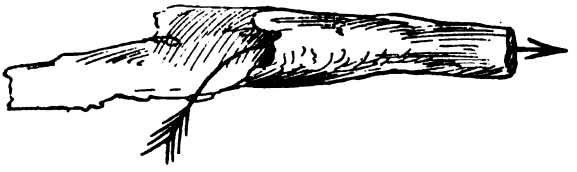
On further examination diaphragm found paralysed, with reversed movements at epigastrium. Knee-jerks absent. Copious moist râles all over lungs. 11 P.M.—Has improved; pulse regular; less restless; ? slight action of diaphragm.

June 22.—Improvement maintained. 10 P.M.—Less well; more restless; signs of diaphragmatic paralysis more obvious. Pulse more rapid. At 1.30 A.M. next morning child suddenly became very cyanosed, and ceased breathing. All attempts at restoration failed. No post-mortem.

XX.—*An unusual case of Diphtheria of the Air-passages.* By E. W. Goodall, M.D. *Read March* 1, 1895.

ALBERT H., æt. 4, was admitted into the Eastern Hospital on May 18, 1892. There was thick membranous exudation on each tonsil; the fauces were swollen and injected. Pulse regular; colour good; knee-jerks present; temp. 102° F.; otherwise the patient appears very well. No albumen in the urine. The patient remained in the same condition until May 25, except for the fact that albumen appeared in the urine on the 24th, and that the membrane was becoming less thick. On the 25th he began to cough occasionally; the cough was slightly stridulous, but there were no signs whatever of laryngeal obstruction. On the 25th the pulse was 88 per minute and regular. On the 27th at 11 A.M. the child called the nurse and told her he had something on his tongue. This "something," which at first sight looked like one of the little masses seen in the nummulated sputum of phthisis, proved on being floated out in water to be the cast of the trachea shown in Fig. 3. There had been no discom-

Fig. 3.



fort during the expectoration of this cast. Pulse 112. Temperature normal on 21st, 22nd, 23rd, 24th, and 25th; 101·2° on morning of the 26th; and 100·5° on morning of 27th. On that day there was still membrane on the tonsils. The boy was rather sleepy but restless; his colour was good; there was no croup, nor even stridor.

May 28.—After 4 P.M. yesterday the boy coughed up some shreds of membrane, and during the night the cast shown in Fig. 4. There was no dyspnœa during the expulsion of the casts or shreds. This morning the patient's colour is

good, and he appears to be well, though his voice is a little hoarse. No ciliary paralysis; knee-jerks present; temp. 101°. There is albumen (a thick trace) in the urine.

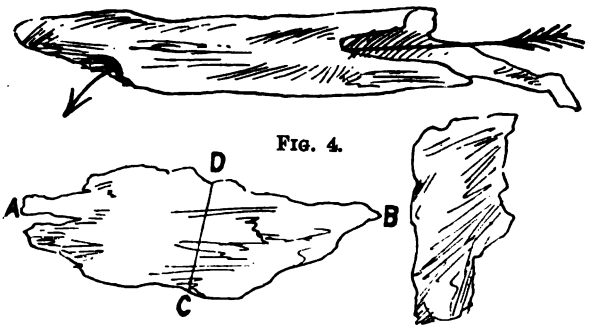


Fig. 4.

From this date up to June 3 the boy coughed up several membranous shreds and casts, the largest of which are shown in the adjoining figures :—

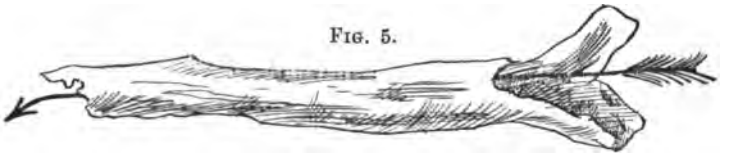


Fig. 5.

Fig. 5, at 1.30 P.M., May 28.
Fig. 6, at 10.30 P.M., May 28.
Fig. 7, at 1.40 P.M., May 29.
Fig. 8, after noon, May 29.
Fig. 9, at 9.40 A.M., May 30.
Fig. 10, at 11 P.M., May 31.
Fig. 11, morning of June 1.

On the evening of May 30 a piece of membrane one inch long was coughed up.

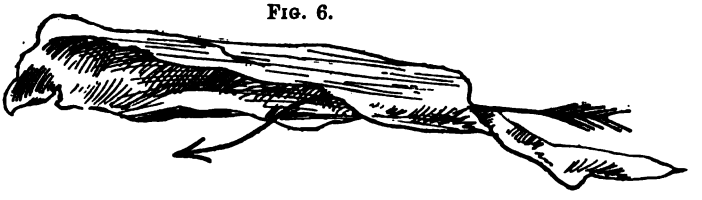
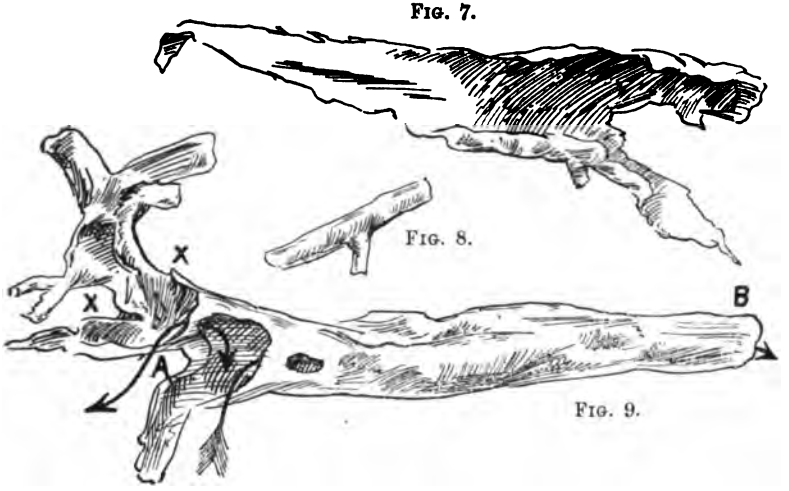


Fig. 6.

From June 1 to the morning of June 3 a cylindrical cast of the trachea 1 inch in length, and several small membranous shreds (one of which was blood-stained) were coughed up.

Fig. 7.



The largest of these casts and shreds was 4 inches in length. Fig. 7 appeared to be a cast of the bifurcation of the trachea. Fig. 8 was a very tough cast of a bronchus. Fig. 9 was a cast of the trachea, two main bronchi, and apparently some of the primary divisions of one of the bronchi; at x x the cast was cylindrical, and the lower part was invagi-

Fig. 11.

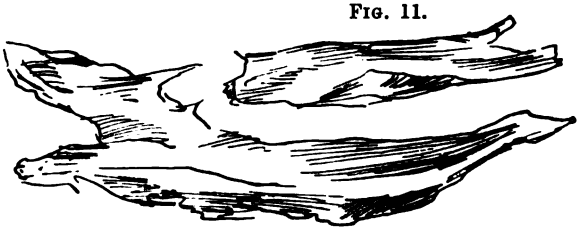


Fig. 10.

nated into it (one division of first bifurcation of bronchus). The invaginated portion was white and thin but tough; the tracheal portion was white and thick.

All these casts were brought up as rounded pellets, and had to be floated out in water to show their form.

There was distressing cough during the expectoration of the casts shown in Figs. 5 and 6; for five minutes before the expulsion of that shown in Fig. 9, the boy had troublesome cough and some cyanosis. The other portions were, however, brought up after very slight coughing, and without any dyspnœa. During the intervals between the occasions when membrane was coughed up, the child had no dyspnœa or cyanosis at all, nor did he seem to be particularly ill. There was never croup. It was noted on May 30 that he was getting weaker, and the pulse-rate was 128. On June 7 paresis of the lower extremities was noted, and next day the knee-jerks (fairly well marked up to that date) were observed to be slight. On the morning of the 15th there was a slight attack of heart failure, and on this date the voice became nasal. On the 18th there was partial paralysis of the ciliary muscles; the patient could stand, but not walk, on account of ataxia. The knee-jerks gradually disappeared, and were not to be obtained on June 29. They were still absent on July 15; no further note was made about them. By July 20 the ciliary paralysis had disappeared, but there was still a slight nasal voice. The patient, however, was quite well on July 27, when he left the hospital.

There had been much albumen in the urine on May 31; gradually the amount lessened until the urine was free from albumen on June 27; after that date it was normal, save on two occasions when there was a faint trace. A little deposit could still be seen on the tonsils on June 6, three days after the last shred of membrane had been coughed up. The temperature was normal after June 3.

The interest of this case lies in the number and size of the expectorated casts, combined with the utter absence of any signs of obstruction to the air-passages. It seems as if the membranous formation, starting in the fauces, missed the larynx in spreading to the trachea. Cases of croup in diphtheria, whether tracheotomised or not, in which casts of the larynx, trachea, and bronchi are coughed up, are of course common enough, but cases such as the one just related are uncommon. I have only met with four since I went to the Eastern Hospital at the beginning of 1892. The first was the present case. The second was that of a girl æt. 4, who was admitted to the hospital on May 30, 1892, with membrane on both tonsils; there was a slightly stridulous cough, no dyspnœa, colour good. The next day she coughed up without difficulty three membranous shreds,

not cylindrical, from 2 to 2½ inches long and ½ to ¾ inch broad. On June 1 there was slight stridor, and another shred 1¾ by ¾ inch was coughed up; no more membrane was coughed up, though slight stridor existed till June 4. There was albuminuria from admission to June 25. On June 25 symptoms of paralysis appeared, nasal voice, paraplegia, irregular heart. The patient recovered, and was discharged well on July 27.

The third case was a boy æt. 4, a very rickety child; admitted May 2, 1892, with membrane on both tonsils. On May 9 he had a cough which was altered in character, but there was no stridor or laryngeal obstruction. On May 12 he coughed up a piece of membrane, partly a hollow cylinder, 1½ by ⅜ inch in size. On May 13 he coughed up another piece 2 inches in length, and not cylindrical. There was albuminuria from May 9 to July 1. About May 25 symptoms of paralysis appeared in the legs; subsequently the paralysis became generalised and severe, but the child recovered, and was discharged on September 13 quite well.

The fourth case was a young woman æt. 18, who was admitted into hospital on July 26, 1894, with severe faucial and nasal diphtheria. She brought up numerous tracheal casts showing the marks of the rings of the trachea. There was no symptom of obstruction to the air-passages. The urine contained much albumen. She died on July 31 of the severity of the disease. The Klebs-Löffler bacillus was found in the faucial membrane.

For the pen-and-ink sketches of the casts in the first case I am indebted to Mr. H. E. Mann, who was assistant medical officer at the time.

XXI.—*A case of Malformation of the Heart with Hæmophilia.* By Lee Dickinson, M.D. *Read March* 1, 1895.

THIS case is brought forward in the hope of obtaining opinions, by which my own may be directed, as to the relationship between the two diseases which it combines.

Louisa F., æt. 8, is the second child of a healthy mother who gives no family history of hæmophilia or other disease.

She has been known to have heart disease since the age of two, but had no discomfort referable to this till within the last three years. During this time she has been liable to attacks of dyspnœa and præcordial pain with violent action of the heart on exertion or exposure to cold; these attacks appear to be increasing in severity and frequency.

She has always bruised readily. When she was about a year old a very large bruise appeared spontaneously upon the forehead. A tooth was extracted two years ago, and the attendant hæmorrhage was of a very alarming character. For the last four years she has been subject to frequent epistaxis, an attack of which in July, 1892, necessitated plugging the nose. In February, 1893, she was admitted into St. George's Hospital, being stated to have coughed up a large quantity of blood. It was soon found that the blood had come from the nose, from which there was a constant oozing for a couple of days, culminating in profuse epistaxis with vomiting of swallowed blood, whereupon the child became decidedly blanched. The hæmorrhage ceased after the liberal use of ice and plugging the nostrils, and did not recur. The naso-pharynx was examined and found to be free from disease. The child rapidly recovered from the loss of blood. Since then her nose has bled on several occasions—so freely in the spring of 1894 that a medical man was called in; again in the autumn for four hours continuously, after which she was admitted into the Victoria Hospital for Children; and probably at the beginning of December, when she was stated to have had an attack of blood-spitting. These various and repeated hæmorrhages seem to justify the diagnosis of hæmophilia.

The child is fairly well grown and intelligent, and well formed externally except for some imperfection of

the sternum and ptosis of the left eyelid. The irides differ from one another slightly in colour. She has a florid complexion, of a red, not blue, tint. There is no clubbing of the fingers or toes. The lungs and abdominal organs appear normal. The heart appears natural in size and position, but over the whole chest is audible a loud, long, rough systolic murmur, louder to the left than to the right of the sternum, generally loudest at the apex of the heart, where is a thrill synchronous with the murmur. The same thrill can be felt in the episternal notch. In the neck there is distinct venous pulsation. From these signs and from the history it is fairly clear that there is congenital heart disease, and that probably the chief defect is of the septum ventriculorum, with perhaps pulmonary stenosis and secondary tricuspid incompetence.

Remarks.—It is an old observation that the subjects of cardiac cyanosis are liable to hæmorrhage. In 1811 Nasse drew attention to this liability, which he attributed to deficient coagulability of the blood owing to its venosity, remarking that for the same reason the whale bleeds to death from slight harpoon wounds.* Similar reflections were uttered a few years later by Meckel after a survey of 77 cases of malformation of the heart.† Both Nasse and Meckel imagined a relationship between cyanosis and hæmophilia on the ground of imperfect development of the blood. Afterwards came Schönlein, whose teaching that hæmophilia was connected with malformation of the heart was apparently founded as much upon the hæmorrhagic tendency in cyanosis as upon the state of the heart in hæmophilia.‡ During the last half-century these views as to the relations of hæmophilia have become obsolete without having ever been fairly discredited. I think, however, that the frequency of hæmorrhages, even of a serious degree, in congenital heart disease, will not be denied by anyone who has in mind a sufficient number of cases. Mention of it is not omitted in the classical work of Dr. Peacock nor in the valuable monograph of Rauchfuss.

* *Reil's Archiv f. die Physiologie*, Bd. x, p. 266. The fact of the enormous whale being killed by the puny harpoon may be accounted for in a more probable manner. "The non-valvular structure of the veins in the Cetacea, and the pressure of the sea-water at the depths at which they retreat when harpooned, explain the profuse and deadly hæmorrhage which follows a wound that in other Mammalia would be by no means fatal" (*Penny Cyclopædia*, vol. xxvii, p. 285).

† *Deutsches Archiv f. die Physiologie*, 1815, Bd. i, p. 248; and 1816, Bd. ii, p. 138.

‡ Schönlein insisted upon the great inclination to hæmorrhage in cardiac cyanosis, *Pathologie und Therapie*, 1837, Bd. ii, p. 55.

In many cases, no doubt, the hæmorrhages may be wholly or partly explained by venous congestion or by mere venosity of blood; but these conditions can have little or nothing to do with an hæmorrhagic tendency in cases, like the one brought forward, in which there is neither clubbing of the extremities nor cyanosis. Here at least is an unequivocal diathesis.

In a certain number of post-mortem examinations upon persons dead of hæmophilia, some malformation of the heart, or more frequently of the vessels, has been discovered. Apart from this association, it is possible that the two diseases are related in a developmental peculiarity of the blood. It appears from several independent observations that the blood in congenital heart disease, the cyanosis of which has hitherto been so difficult to explain, is in a state of concentration. Lloyd Jones, who took the specific gravity of the blood in five cases, found it very high, ranging from 1061 to 1072.* Banholzer found in a case of pulmonary stenosis that the specific gravity was 1071·8, while the number of red corpuscles in a cubic millimetre was 9,447,000. He refers to two similar cases, in one of which the number was 8,000,000, in the other 8,900,000.†

Quite recently this subject has been dealt with by Dr. G. A. Gibson,‡ who contributes two observations to the same effect and refers to three others.

As to the condition of the blood in hæmophilia, it will probably be admitted that the natural aspect of the majority of bleeders is sanguine or even plethoric. It has not yet been proved that there is an absolute excess of red corpuscles, though this is highly probable from the fact that they have been found in great relative excess by certain careful observers, *e. g.* Finger§ who examined the blood in a well-marked case of hæmophilia repeatedly during a period of nearly five weeks, and Assmann,‖ who made similar examinations in another case during fourteen weeks. The latter found on an average 1500 red corpuscles to one white.

* *Journ. of Physiology*, vol. xii, p. 327.

† *Centralb. f. innere Medicin*, June 9, 1894.

‡ *Lancet*, Jan. 5, 1895. It is only right to state that Dr. Gibson considers the increase in the number of corpuscles to be secondary to venous stasis.

§ Quoted by Grandidier, *Schmidt's Jahrbücher*, cxvii, p. 334.

‖ *Die Haemophilie*, Inaug.-Dissert., Berlin, 1869, p. 31.

XXII.—*Gangrenous Umbilical Hernia: immediate resection of gut, and union by Murphy's button: recovery.* By GILBERT BARLING. *Read March* 1, 1895.

A FEMALE, æt. 49, was admitted to the Birmingham General Hospital on the afternoon of December 21, 1894, with a strangulated umbilical hernia.

Until December 17 the hernia had been reducible, but at 4 P.M. that day the patient had severe pain at the umbilicus, and found she could not reduce the hernia; she vomited at 6 P.M.

The vomit began to be offensive in the evening of the 18th; vomiting and pain continued until the morning of December 21, when both ceased. The only treatment ordered before admission to the hospital was an enema, administered on December 20, and which caused a fairly copious motion.

On admission, the patient, a stout, anæmic-looking woman, had a fairly placid face; her pulse was 90, temperature 97°. She had not vomited for some hours, and was not in pain. The hernia was about the size of half a large orange, flattened out, the skin over and around it was considerably indurated and somewhat reddened. The central portion was resonant. No attempt at reduction was made.

Operation.—Incision over the middle of the hernia showed slightly strangulated and indurated omentum adherent nearly all over. This was separated and removed, liberating a quantity of deeply blood-stained fluid, behind which was a loop of small intestine. The gut was tense, of red-blue colour, and presenting in places a grey appearance, as though from sloughing tissue in the inner coats. The constriction was by a band crossing the lower margin of the hernial opening; the band was rather flat, about ¼ inch wide, and nipped the gut with great severity. On severing it a line of dead white gangrene was found at the point of constriction on the entering intestine. The intestine was brought well outside the abdomen and carefully packed round with sponges, and the constricted portion, about 5 inches, with a V-shaped piece of mesentery, was removed

with scissors, the gut being clamped by assistant's fingers. The incision was carried through healthy intestine about an inch on each side beyond the constriction. There was rather troublesome bleeding from the divided mesentery. A running suture was now inserted in each end of the intestine, beginning opposite the mesentery and ending at that point, securing approximation of the two layers of the mesentery by a return over-stitch, so that the gut was not denuded of peritoneum. One half of the button was then inserted into each piece of intestine, the running suture tightened around its shank, and the two portions fastened together with moderate firmness. The approximation seemed very secure, and no other sutures were used. The divided edges of the mesentery slightly overlapped, and were not sutured. The hernial orifice had to be enlarged to permit the return of the bowel. The wound was entirely closed.

After-treatment and progress.—Feeding was entirely by enemata until December 24, when a little milk was given by the mouth, and from this time the enemata were gradually left off as food by the mouth was increased. There was no vomiting at any time; flatus was passed on the 22nd, and the abdomen throughout was flaccid and free from tenderness. The pulse ran to 100 or rather more for a month after the operation, and at first was extremely hard. The bowels were evacuated by enema on the eighth day after operation, and though an action was obtained in this way nearly every day, the button was not passed until the twenty-fourth day after operation. It contained a ring of necrosed tissue, which I show here.

The only complaint made by the patient was of occasional pain, "like indigestion," felt in the upper part of the abdomen, and attributed by her to taking milk. Patient is now (February 28, 1895) well in all respects.

Comments.—The sudden cessation of pain and vomiting when they had existed over three days, the hernia not having been reduced, suggested gangrene, although the condition of the hernia itself suggested that it might simply be an inflamed irreducible hernia.

Resection of gut and immediate union was determined on, rather than the formation of an artificial anus, because of the patient's good general condition, and because it appeared that an artificial opening would be high up the small intestine, a most undesirable position.

The button used measured barely one inch in diameter.

An attempt was made to employ a size larger, but it would not easily enter the intestine.

The mere insertion and securing of the button and approximation of the intestine was very easy, much easier than the approximation, for instance, by Senn's plates.

The length of time which elapsed before the button was passed was probably due to the inactivity of the intestine only, the woman being always constipated. For this reason she was allowed to get up on the twenty-second day, the button being passed two days later.

The patient's future is a matter of great interest.

The point on which we require information now is as to the continued patency of the united intestine. Will the circle of union produce a stricture. If it does not, this method of union has a great advantage over many others, for it is a matter of rather frequent experience that the communications made between various parts of the alimentary tract have a tendency to contract and may become completely closed.

XXIII.—*A case of Hysterical Contracture of the Legs of two years' duration successfully treated.* By Hector W. G. Mackenzie, M.D. *Read March* 1, 1895.

THE patient whose case I am about to relate is a young woman who, at the time she came under my care for the contracted condition of her legs to be presently described, was 23 years of age. I had first seen her some three years previously at the Brompton Hospital, where she attended on account of emaciation and pain in the chest, which, it was feared, pointed to consumption. Her symptoms, however, gave little support to this idea, and finding nothing abnormal on examination of the chest, I thought it better that she should be treated at a general hospital, and arranged that she should be admitted under my care to the Royal Free Hospital.

The following is an account of her condition on that occasion, recorded in January, 1890. M. W., æt. 20, a single woman. Anæmic and extremely emaciated. Weight, 5 st. 9 lbs. Arms and legs very thin. Abdomen retracted and boat-shaped, so that there seemed to be nothing between the skin and the vertebræ, its surface mottled from the constant application of poultices, and more or less tender all over, but especially so in the left iliac region. Thorax long and contracted in its lower part. No abnormal physical signs in the chest. No evidence of disease of any of the abdominal organs. Urine normal. Until the age of thirteen the patient had enjoyed good health. Then she had an attack of bronchitis which left a liability to colds from which she suffered in subsequent winters. Her bowels apparently had always been obstinate, but about the age of eighteen constipation became more troublesome, and it was not uncommon for it to last ten days or a fortnight. About the same time she began to suffer from pain immediately or soon after food, and from attacks of vomiting which generally gave relief to the pain. These symptoms persisted, and the patient steadily lost appetite, strength, and flesh. Complete amenorrhœa had existed for eighteen months when she came under treatment.

The subsequent history leaves no doubt in my mind that her troubles were at this time, as they were in their later

developments, purely functional. With my then knowledge of the case, I must say I was inclined to believe that she had gastric ulcer. The treatment adopted was to regulate the action of the bowels by enemata and laxatives, and to limit the food to milk and chicken broth. While in the hospital the patient continued to complain of pain, but there was no sickness. The pain was relieved by the local application of blisters. At the end of a fortnight the patient left the hospital at her own request. She attended after this as an out-patient for a month or so, and I then lost sight of her for a time.

It appears that soon after she ceased her attendance she had some disappointment, some love affair I believe, following on which she lost power in her legs, and had to be taken about in a bath chair. She took to her bed in the following winter. The legs soon became contracted and remained so, with the result that the patient was quite bedridden.

I was informed of her condition in the autumn of 1892, and agreed to admit her once more to the Royal Free Hospital, which she re-entered on October 22 of that year.

She was now twenty-three years of age. Her condition was a very pitiable one. She was even more emaciated than on the previous occasion, her weight being now reduced to 5 stone. It was impossible for her to extend the legs on the thighs at a greater angle than a right angle, and she was quite unable to leave her bed.

On examination it was found that although the legs could not be extended beyond the right angle they could be flexed up to the normal limit. The flexors were in a condition of tonic contraction. The movements at the hips were normal, except that the psoas appeared tighter than natural. The feet were over-extended, but the movements at the ankles and toes were normal.

I may add here that on several occasions the patient was put under the deepest anæsthesia without in the slightest degree relaxing the tension of the flexor muscles, and it seemed certain that any attempt at forcible extension could only result in rupturing the muscles or their tendons.

The knee-jerks could not be elicited. The muscles of the thighs and legs were much atrophied, while the flexor tendons were hard and rigid. The electrical reactions of the muscles were normal.

The following measurements illustrate the degree of attenuation :—The greatest circumference of the calves was

8¼ inches, that of the right thigh 3 inches above the patella was 9⅜ inches, that of the left 9⅛. The abdomen at the umbilicus measured 22 inches, and the thorax at the nipple level 29 inches.

There was no sign of disease in the chest.

The skin of the abdomen was, as before, much pigmented. The abdominal wall was extremely depressed, and the bony prominences stood up very plainly. There was no sign of enlargement of liver or spleen.

The urine was normal.

The mental condition was one of depression. The patient talked in a low monotonous tone—always about her own ailments. She wept without assignable cause. She seemed, however, anxious to have her legs made right.

There was no anæsthesia or analgesia. The sole of the left foot, especially about the ball of the great toe, was hyperæsthetic. The patient complained of tenderness over the lumbar and lower dorsal vertebræ, over the posterior iliac spines, along the iliac crests, in the groins, and over the abdomen generally. The special senses were all normal. She was continually, however, shutting her eyes, and said she could not bear a bright light. The catamenia had now been absent for four years.

Her appetite was very bad. Food, she said, tasted like clots of blood. She stated that she had occasionally had vomiting, spitting of blood, and pain in the epigastrium since leaving the hospital. The pain had been relieved from time to time by the application of blisters. Once she had spat enough blood to soak a handkerchief.

She had sometimes experienced pain and difficulty in micturition, and sometimes at night had wetted the bed.

Since her legs had become affected she had had three fits. The first had occurred two years before, and had lasted nearly four hours. During the fit her hands were clenched and her mouth was drawn to one side, but she had never bitten her tongue.

It may be added that she resided with her parents. Her mother was extremely solicitous about her, pitied her to a degree, and humoured her in everything. One sister suffered with incontinence of urine, and another from inflammation of the bladder.

Putting all the facts together, I could come to no other conclusion but that the case was functional, one of anorexia nervosa combined with contracture of the flexors of the legs.

As far as I could see, only a functional origin could explain a limited contracture of this kind, without more loss of power than was likely to accompany simple wasting and disuse of the muscles. If the leg affection was functional, it was only natural to believe that the gastric trouble was of the same nature. The mode of onset of the contracture was exactly that which one would expect in such a case. There were, moreover, fond and sympathising relations to aggravate and perpetuate all the symptoms. Although, however, the disease was functional in its origin, it seemed certain that organic changes in the muscles had supervened. I was aware of the belief of Charcot, to which I shall refer later on, that when contracture has long existed it becomes incurable. It seemed at first sight that nothing would straighten the limbs except tenotomy, and with the extremely wasted condition of the muscles there was a very poor chance of tenotomy proving a lasting benefit. The absence of fibrous adhesions in the knee-joints was a favorable point. I felt that the first matter of importance was to improve the muscular condition, and when that was accomplished, I hoped that by carefully graduated exercise the contracted muscles would relax and the limbs straighten themselves. To cure the morbid mental condition it was obviously essential to cut off the patient from the injudicious attentions and sympathies of her relations, and accordingly all visitors and letters were strictly prohibited. I thought the case might quite well be treated in the general ward of the hospital by massage and a liberal dietary. Here I should like to remark that I do not believe that it is ever essential that an hysterical neurasthenic patient should be isolated in a room by herself. No doubt it is an advantage in some cases, but I consider, that if communication with friends is stopped, cases can be quite successfully treated in the general wards. This is a very important matter, for a separate room is often unattainable and its employment adds greatly to the cost of treatment.

I have myself obtained most satisfactory results in the general wards, even in cases where failure has attended Weir Mitchell treatment strictly carried out by complete isolation in a separate room.

Massage was commenced on November 16. It was ordered for half an hour twice a day, the legs to have ten minutes each, the abdomen five minutes, and the arms five minutes. Her food was supplemented by the addition of five ounces of milk between each meal.

The massage was increased to three quarters of an hour twice a day on December 3, and to an hour a day on December 9.

Her diet was also soon made an extremely liberal one, so that in addition to substantial meals she was taking three pints of milk and a pint of strong soup in the twenty-four hours. A little difficulty arose in consequence of her regurgitating small quantities of food, amounting to about half a pint in the twenty-four hours, but this stopped when Dr. Rendel, who was then the house physician, threatened that if it continued the patient would have no visitors for Christmas.

At the end of five weeks the patient had gained a stone in weight. Her calves now measured 9 inches, her thighs 10¼ inches, and her abdomen 24½ inches in circumference. She had become much more cheerful and much more inclined to occupy herself and to take interest in her surronndings. An attempt was now made to get her out of bed and to make her use her legs a little. In extreme extension the legs now made with the thighs an obtuse angle of 130°. A mechanical contrivance, consisting of a high rail carried on wheels in the shape of a pulpit, proved to be very useful. The patient stood in the interior, supported herself on the rail with her arms, and pushed herself and "the pulpit" along.

On January 6 my colleague, Mr. Battle, kindly saw the patient. He was of opinion that there was some pathological change in the muscles, as the legs could not be straightened under chloroform. He suggested weight extension, but thought there might be liability to suppuration. Failing extension, he advocated tenotomy.

However, I felt hopeful, as the patient had improved so much up to this point, that by continuing the treatment already pursued she would make a complete recovery.

The massage was continued. The patient was made to go on with her attempts at walking exercise.

The contracture gradually but surely gave way, and by the beginning of February the legs could be straightened except for an angle of about 20°.

The calves had by this time increased 2 inches, and the thighs 4 inches in circumference.

The patient now weighed 7 st., a gain of 2 st.

By the middle of February she was able to walk alone.

About this time she was somewhat alarmed by the legs becoming swollen and painful when she walked, but that trouble soon passed away.

She was discharged from the hospital on the 17th of March, 1893, with her legs straight and perfectly able to walk. She weighed 7 st. 11 lbs., showing a gain of nearly 3 st. during her treatment in the hospital. She had changed from a condition of melancholy depression and helplessness to one of cheerfulness and activity, and was extremely grateful for the alteration for the better. Her further progress has been completely satisfactory. She has continued to put on flesh, so that now she weighs nearly 11 st., and looks a healthy, well-nourished woman. She leads an active life, and is a useful member of society. She has but one trouble, and that is occasional attacks of vomiting. For this, experience has taught her that the passage of an œsophageal bougie acts as a certain cure, and from time to time she attends to have this done.

I have already incidentally referred to the diagnosis. Of great importance was the mode of onset. There had been some mental trouble, after which the patient had taken to her bed and the legs had gradually become contracted. There was nothing to suggest that there had been neuritis. There was no more or less sudden loss of power as in the case of myelitis, nor did the condition at all resemble that of paraplegia from myelitis or caries of the vertebræ. There was nothing to point to the existence of sclerosis. It is worth while again mentioning that the muscles were all much wasted, that the loss of power in them was only commensurate with the atrophy, that the flexors of the legs were extremely contracted, that the legs could not be extended beyond a right angle, but could be flexed to the normal limit, and that the contracture remained unaltered under the deepest anæsthesia.

Cases of hysterical contracture such as this are not at all common. I have never myself seen an exactly similar case. It is conceivable, however, that, being considered incurable, they are not often brought to a hospital, and are more likely to come under the notice of general practitioners or the medical officers of infirmaries. But even those with the largest experience speak of such cases as rare, and it must be borne in mind that the form of contracture in this case was not the usual one met with in hysteria. "The position of the legs," writes Dr. Gowers, "in the paraplegic form of hysterical contracture is rigid extension. The flexor spasm occasionally seen in organic disease is scarcely ever met with in hysteria." This opinion is supported by that of Charcot, according to

whom the permanent flexion of the thigh and of the leg is a condition really exceptional, the ordinary type being rigid extension, the thigh strongly extended upon the pelvis, the leg on the thigh, the foot in the position of equino-varus, and the adductor muscles strongly contracted.

With regard to prognosis Gowers states that severe forms may last for years, the contracted muscles becoming permanently shortened. Charcot remarks that in all cases if cure takes place it will take place suddenly, but sometimes the contracture persists and becomes an incurable infirmity. It is obvious where the muscles have become shortened, and under the deepest anæsthesia no extension is produced, sudden cure is a physical impossibility. The condition of my patient, according to Charcot, might therefore well be thought incurable.

Weir Mitchell, whose opinion is especially valuable, gives it as his experience that the chronic spasms of women with hysteria are, perhaps, among the most obstinately unmanageable of all the graver symptoms of this disorder. "I do not see," he says, "abrupt recoveries. We earn our triumphs, as a rule, by intelligent and patient care."

As regards treatment, I have little to add. At first sight it seemed certain that the muscles were permanently shortened, and that nothing but tenotomy would permit the legs to become straight. As I have said, I am very doubtful whether any benefit would have arisen from performing tenotomy when the muscles were so much wasted. When the general nutrition was improved and the muscles redeveloped, it was found that tenotomy was not required, the legs gradually straightening themselves with use. According to Weir Mitchell, section of tendons in hysterical contractions is as a rule unadvisable. He speaks of one case where in despair he cut several tendons, and the contractions returned with violence.

In view of the generally received opinion that cases of this kind are usually incurable or intractable, it is very satisfactory to be able to record that in so well-marked and long-standing a case as that of my patient ordinary methods of treatment have proved sufficient to establish a complete and permanent cure.

XXIV.—*A case of Cerebral Tumour (Sarcoma) following Injury.* By HENRY HANDFORD, M.D. *Read March* 8, 1895.

IT is well known that blows upon the head are very frequently followed by headache, pains, and other nervous phenomena, which may be exceedingly troublesome and persistent, but which almost invariably get quite well in the end. And in the more severe injuries, where recovery does follow at all, it is frequently remarkably complete and enduring.

On the other hand, we occasionally meet with cases hardly distinguishable at the beginning from the first group, where the patient dies in a few months of cerebral tumour. The importance of distinguishing between these two classes of cases is my excuse for bringing the present case before this Society.

The patient whose history I am about to give was sent to the hospital with a diagnosis of hysteria. A previous and almost exactly similar case, of which, with your permission, I will give a short *résumé*, was sent to the surgical wards on account of paralysis of the leg, supposed to be due to an injury to the hip, without any suspicion of cerebral disease.

In the first case* a coal-miner was crushed by a fall of coal. There was no scalp wound; he did not lose consciousness, and with the assistance of two companions he walked a short distance to his home. For the first fortnight he was able to get up for a few minutes daily, but required assistance in getting in and out of bed. About three weeks after the accident the left leg began to "jump," and a week later a feeling of "pins and needles" gradually spread up the leg to the side of the trunk, and to the left arm, followed by pain and gradual loss of power. Common sensation on the left side was diminished. The pupils were unequal, and while in the hospital optic neuritis commenced and advanced rapidly. The paralysis gradually advanced, and spread to the right leg, the mental faculties became more and more blunted, and the patient died, comatose, eighteen weeks after the accident, without at any time having had fits or muscular spasm. At the post-mortem there was no sign of injury to the bones of the skull, but a dark-coloured tumour was dis-

* *Brain*, 1891, p. 269.

covered in the cortex occupying the upper end of the right ascending frontal convolution, and the posterior extremity of the superior frontal, bulging slightly, firm to the touch, and about 1⅓ inches by 1 inch in size. On the median aspect it occupied the greater part of the paracentral lobule.

On microscopic examination of the tumour, which had been hardened in Müller's fluid, it was found to be an angio-sarcoma. There was an abundance of small round cells, some small spindle cells, and a few large round oval nucleated cells. The intercellular substance was scanty, granular, and in some places fibrillated. The blood-vessels were very numerous, of large size, and with greatly thickened hyaline walls. There were numerous small hæmorrhages and several areas of degeneration.

In all probability the primary loss of power in the left leg was due to a hæmorrhage caused by the accident, and the subsequent paralysis to the growth of the tumour in the injured tissues.

The next case bears such a very close resemblance to the one just related that I was enabled, by a recollection of the former one, to come to an exact diagnosis as to the presence of a tumour and its probable nature, but the material did not exist for a very exact localisation.

L. M., æt. 43, was admitted August 16, 1894, complaining of "pain in the head, and sometimes of loss of use in the legs."

He has been employed as cooper at a brewery for thirty-one years, but his wife says he is exceptionally temperate. He has been a strong, healthy man.

Four months before admission, while getting out of a cart, he caught his foot and fell six feet on to the pavement, striking especially the right side of his forehead, and injuring the right shoulder. He did not lose consciousness, and walked home. Next day he seemed quite well, except for the pain in the shoulder and over the right eye. He did not vomit at the time of the accident, and has not done so at any time during the illness. No bruising was detected upon the head, but "the veins stood out on the right temple." The headache has persisted ever since, but now is slightly better; it has been very severe. He has had no fits of any kind. After about a fortnight he began to be rather despondent, especially about his right arm, as he did not regain much use in it.

He gradually became worse, and for the six or seven weeks before admission was chiefly confined to bed. He could

walk with assistance, but had very little power either in the arms or legs. He has often wetted the bed, and occasionally has had retention of urine for a day, but has never required a catheter to be passed. He has passed his motions into the bed occasionally. For the last three weeks he has been very sleepy. He was sent into the hospital with a diagnosis of "hysteria"!

When first seen he lay in bed, never speaking except in answer to questions, and then generally in monosyllables and after an interval of nearly a minute. His face was flushed, he yawned frequently, and seemed oppressed. He could move all his limbs freely, but they seemed weak. His articulation was quite good.

August 23.—He lies like a log in bed, and emptied his bladder and rectum into the bed; there is no dribbling. He waits about sixty seconds before answering a question. His breathing is noiseless and slow. He can move both legs freely. The knee-jerks are increased but equal. There is no loss of sensation in the legs, but the skin is rather cold.

The right arm can be moved freely, and sensation seems normal.

The left arm is colder than the right, and the muscles seem wasted. Common sensation is not lost, but is much blunted. The grip of the left hand is very feeble. He can extend the fingers, but not the elbow, though he can flex it. He can put the left arm on the forehead with difficulty, but not on the top of the head; and when placed in the latter position it is only with great difficulty that he can remove it. He does not move the lips or facial muscles when told. There is probably some weakness of the facial muscles on the left side, as when the whiskers are pulled on the right side the muscles of expression on that side contract; but when they are pulled on the left he evidently feels the pain, but there is no muscular contraction. The pupils are equal, moderately dilated, and do not contract to light. It is impossible to fix his attention sufficiently to ascertain whether they react to accommodation. There is very extensive optic neuritis in both eyes, with numerous small hæmorrhages and abundant exudation of lymph.

His pulse varied in speed from 70 to 104 per minute, but was otherwise regular, full, and soft.

August 27.—Paralysis of the left arm and leg is now nearly complete. He was noticed to contract both halves of the occipito-frontalis equally on one occasion; but the

muscles of the lower part of the face on the left side are weaker. He is only semi-conscious, and takes no notice of his surroundings. Tapping the head over the squamous portion of the right temporal bone causes the right facial muscles to contract violently and remain in spasm for many seconds. Tapping the left side only produces slight left frown. The eyes frequently deviate conjugately to the right.

A diagnosis of tumour had been arrived at; and an operation was thought advisable as a last resource, but without much expectation of cure. The localisation of the tumour was difficult. The absence of any scar prevented the injury being used as a guide to the site of operation. The complete absence of epileptiform fits or localised muscular spasms suggested that the lesion was subcortical. The want of any indication of paralysis of the cranial nerves or of interference with the medulla seemed to indicate that the lesion was not at the base. The severity of the general symptoms before there was any persistent local paralysis pointed to an extensive lesion outside the direct motor tract; while the gradual creeping onset of paralysis, affecting finally the left face, arm, and leg, led us to believe that the lesion was in close proximity to the motor tract, and on the right side of the brain. The latter point was borne out by the history of the original injury. It was much more difficult to determine whether to make the exploration in front, behind, or below the motor area. The patient's mental faculties were too much blunted, even on admission, for any accurate examination of his special senses to be made. But he could certainly hear and see, for he answered questions (though imperfectly), and could count fingers and recognise faces. Therefore it was thought less probable that the injury was in the occipital or temporo-sphenoidal lobes, and it was decided to trephine immediately in front of the motor area, especially as the history of the accident pointed to that region as the site of the blow.

Accordingly on August 27 the operation was most ably and skilfully performed by my surgical colleague, Mr. Anderson. The brain bulged excessively into the opening, but no sign of tumour could be detected, though the brain substance was explored to a depth of about 2 inches. A second opening was made lower down with a similar want of success, though the brain substance looked slightly discoloured. No further exploration was advised, as the patient, who had been getting rapidly worse from the date of the last observations

until the time of the operation, was in an extremely prostrate condition. Death followed in about twelve hours, and at the necropsy no fracture or other injury was found in the calvarium. The dura mater was normal. There was a considerable excess of clear fluid in the subarachnoid space and in the lateral ventricles, which were dilated. The membranes showed no abnormality, except some darkening and opacity over the right upper temporo-sphenoidal lobule, the centre of the second or middle temporo-sphenoidal lobule, and a small portion of the adjacent parietal lobe.

On a horizontal section of the brain being made, a mass about 4 by 1½ to 1¾ inches was seen outside and behind the right corpus striatum. It was harder in texture than the surrounding brain substance, ill-defined at the margins, dark red, and mottled in colour. It evidently contained much blood-pigment. The naked-eye resemblance of the growth to that in the former case was complete. Wishing to obtain an independent pathological opinion as to the nature of the growth, I sent a portion to the new Clinical Research Association without informing them of the opinion I had formed in the previous case. The report, dated September 21, runs as follows:—"The tumour is a vascular sarcoma composed of round and spindle cells." On examining the specimens which are under the microscopes in the adjoining room, I find that, in addition to the round cells with large single nuclei, and the spindle cells, there is an abundant intercellular substance which is in most places granular, and in some finely fibrillated. In other portions of the section there is a coarse stroma, the majority of the meshes of which are filled with well-defined, closely-packed red blood-corpuscles. The walls of the blood-vessels are much thickened. I think there can be no doubt that the growth is a vascular sarcoma or angiosarcoma, and that it has arisen in the damaged tissues injured by the accident described. It is probable that a blood-clot poured out at the time of the accident formed part of the pabulum upon which the tumour has thriven; but the blood seen in the sections appears, on account of the integrity of the corpuscles, to be either part of more recent interstitial extravasations, or part of the vascular sinuses of the tumour, or both.

It is stated that the growth of a tumour in the brain in consequence of an injury to the head is rare, but that in the recorded cases the nature of the tumour has been of very different kinds, including both tubercle and syphiloma.

XXV.—*Cases illustrating the association of Psoriasis with Diabetes Mellitus.* By KARL GRUBE, M.D., of Neuenahr, Germany. *Read March* 8, 1895.

GENTLEMEN,—Will you allow me to bring before you in this paper some observations which I have made on the combination of diabetes mellitus with psoriasis, which to me seem to indicate a connection between these two complaints, and which, I hope, may be of some interest to you?

Both affections are still surrounded by much darkness, and we know very little of their causes and their pathology. Some are of opinion that psoriasis is an affection of the nervous system, and they call it accordingly a "neurosis," which is a very convenient term, but which translated into plain language does not say more than that we have to do with an affection of whose character and causes we know nothing, although it is probably of nervous origin. This view of psoriasis being a neurosis has been strongly expressed by Professor Polotebnoff,* of St. Petersburg, and some years before by Bourdillon † in France.

On the other hand, there are cases of diabetes which we are unable to explain otherwise than by assuming the existence of a primary affection of the nervous system which leads to that disturbance of metabolism generally known as diabetes mellitus. This form of diabetes has been called "neurogenic diabetes," by F. A. Hoffmann, of Leipzig. It is very often observed in young persons, and is not seldom hereditary. The cases in which I observed the combination of psoriasis and diabetes belong mostly to this form.

Before bringing these cases before you I would make some short remarks on what I have found in literature on this combination.

Affections of the skin are not unfrequently observed in diabetes, although I have not been able to find in books treating of diabetes, any remark on the combination of psoriasis with that disease, and I have also failed to find any allusion of this kind in books on diseases of the skin. We must therefore conclude that this combination is not

* *Dermatologisch Studien*, Reihe 2, Heft 5.

† 'Psoriasis et Arthropathies,' *Thèse de Paris*, 1888.

frequent, a conclusion which is confirmed by my own experience, as I have met with it in only five instances out of about 200 cases of diabetes.

But turning to the literature which deals more especially and comprehensively with psoriasis, I have been able to find some mention of the association of the two diseases.

Polotebnoff refers, in his paper on psoriasis, to diabetes mellitus as to one of the "objective disturbances of the nervous system to be observed in psoriasis;" and Nielsen,* in a paper also dealing with psoriasis, remarks that he found in a few cases chronic nephritis and *once diabetes mellitus*. The same author states that psoriasis is frequent among Jews, a fact also known of diabetes.

Gerhardt, in a paper "on the relation of psoriasis to chronic joint-disease,"† also refers to the connection of diabetes and psoriasis.

This is all I have been able to find on the connection between diabetes and psoriasis.

My own cases are as follows:

Case 1.—Gentleman 39 years of age. Father and sister died of diabetes. Patient has been suffering from psoriasis of the trunk and limbs since his boyhood. The affection had disappeared for some years, but had returned five years ago. Diabetes was discovered three years ago, at the same time there was a great increase of the intensity of the psoriasis. The patient said that the affection of the skin was much less troublesome when his general condition was best, an observation which I was able to confirm. The patches became smaller as the condition of the patient showed some improvement. But while the improvement of the psoriasis lasted, the diabetes advanced more and more, and the patient died at last from coma. I was able to make the post-mortem examination. There were only secondary changes to be found in the nervous system, while the pancreas showed no sign of disease, neither by macroscopical nor microscopical examination.

Case 2.—Brother of Case 1, 30 years of age, was quite well till the autumn of 1893, except for psoriasis, especially on the back. At that time sugar was first found in the urine, but it disappeared promptly on restricted diet. Since last spring

* *On Psoriasis*, translated for the Sydenham Society.

† *Berliner klinische Wochenschrift*, Sept. 17, 1894.

the diabetes has become more severe, the patient passing sugar even on very strict diet, and since the autumn of 1894 symptoms of the severe form of diabetes have developed. On the other hand the psoriasis has become much better, in fact it has nearly disappeared.

Case 3.—Gentleman 40 years of age; history of gout. Has symptoms of severe diabetes. There are patches of psoriasis on both legs. The psoriasis is said to have existed for many years, and to have been very troublesome till the diabetes appeared, when it got ever so much better.

Case 4.—This case is very interesting. Gentleman 55 years of age. The psoriasis began as early as 1865; there were patches on the head and trunk, while the limbs remained free. The affection became so troublesome that the patient, a colonel in the Russian army, had to give up the military service. The diabetes was discovered in 1892, but the patient believes that as early as 1884 he must have been suffering from it, as he had a good deal of thirst at that time. The psoriasis was very bad in 1882 and 1883, but was much better from 1884 till 1890. In 1890 a very severe attack of it set in, which lasted till 1891. At that time the skin affection got somewhat better, but the patient was suffering from general weakness and was losing flesh, which symptoms led to the discovery of sugar in the urine in the spring of 1892. In June of this year the patient, who was then passing 6 per cent. of sugar, was treated for his diabetes at Neuenahr, where a great improvement occurred, for after five weeks he was passing no sugar at all, although he was allowed some starchy food, and his general condition improved accordingly. This improvement of the diabetic symptoms lasted during the following two years. On the other hand the psoriasis became worse again, the skin of the greater part of the body being affected. In the summer of 1894 the patient underwent a treatment for the psoriasis which seems to have done him a great deal of good, but as I learnt by a letter which he has lately written to me, the amount of sugar passed in the urine has increased again since October last, and he has now to be more careful with his diet.

It seems to me that we are justified from these cases in assuming a close relation between the two affections, especially if we consider how the affection of the skin and the diabetes seem to alternate with one another. In this connection it is

interesting to note that Bazin in his 'Leçons théoriques et cliniques sur les affections de la peau de nature arthritique et dartreuse,' 1868, seems to have observed the same alternation between psoriasis and affections of the joints. He says that the former became less pronounced, the more intense the latter were.

I am unable to explain satisfactorily the association of diabetes with psoriasis, but it appears to me that the combination is not a mere coincidence.

XXVI.—*Rupture of the Gall-bladder and Liver produced by violent straining, in a patient suffering from obstructive jaundice; profuse hæmorrhage into the bile-ducts and intestines; death within five days.*

By W. Arbuthnot Lane, M.S. *Read March* 8, 1895.

Mrs. L., æt. 54 years, was admitted into St. John's Hospital, Lewisham, on December 20, 1894. She was seen at a dispensary by Mr. Stokes, of Lee, five weeks previously. He found her to be deeply jaundiced. This seems to have developed more than two months previously, during which time she observed that her motions were clayey and her urine mahogany-coloured. She then came under the care of Mr. Reginald Clarke, of Lee Park, who attended her subsequently. She could not give any history of a previous attack, nor had she before experienced any pain or discomfort in the region of the gall-bladder. Her liver was definitely enlarged, and she had pain about the gall-bladder. Except for the presence of the jaundice she seemed comfortable,—so much so that it was impossible to prevent her going about her household duties.

On the morning of December 16, owing, it is thought, to her having taken an unusually strong purge, she was attacked with profuse diarrhœa, accompanied with very violent and distressing straining. During a severe bearing-down effort she suddenly exclaimed that she had felt a very sharp pain in the region of the gall-bladder, as if something had given way. This increased in severity till it became very violent. Mr. Clarke was sent for, and found that her abdomen, which was naturally flaccid and without much fat in its walls, was distended by a rounded tumour which formed a considerable prominence upon its surface. It occupied the position of the gall-bladder, being connected intimately with the under surface of the liver, which was still further increased in size, and presented a thick, round, hard edge. The pain was relieved by morphia. The diarrhœa continued in a slighter degree, and the motions consisted chiefly of blood in considerable quantity. Blood was passed for several hours, and subsequently the motions were dark instead of the white

pasty colour which existed before this recent attack. This fact of the passage of blood was not told till after the patient's death. On the following day Mr. Leopold Burroughs saw her in consultation with Mr. Clarke, when they found the tumour larger, but the pain was not so intense. At this time the patient's general condition was much worse than it was after the sudden onset of the pain the day before. It was decided that an operation should be performed, and to this the patient consented on the 20th, when she was moved into the hospital for that purpose.

On her admission she was very feeble; her pulse was about 90, very weak and irregular; she was very drowsy, but became quite conscious when aroused. Her temperature was 97°. She did not pass any water for thirteen hours, and then with difficulty. Her urine was mahogany-coloured. Her bowels were opened during the day, and the material passed was dark in colour and very offensive. How far the colour was due to digested blood and how far to some bismuth that had been administered was uncertain, but it is probable from the subsequent history of the case that it was chiefly due to blood.

On the 21st she was much worse. Her temperature rose to 100°. I was asked to see her with the view of operative interference, and I saw her in consultation with Messrs. Clarke and Burroughs in the afternoon. The history being very imperfect, we found it difficult to account for the symptoms which developed so suddenly except on the supposition that a stone which had occupied the common hepatic duct had passed on and become impacted in the common bile-ducts and so caused the very acute distension of the gall-bladder—a condition which occurs but rarely under such circumstances. Although we felt that she was practically moribund, we hoped that by rapidly operating and draining the gall-bladder we might possibly give her a chance of recovery. We therefore exposed the gall-bladder by an incision over it. The tumour protruded at once through the wound, when it was felt to be firm and inelastic like a soft growth. On incising it its contents were found to be coagulated blood-clot, more than three-quarters of a pint of which was turned out. It then became obvious that not only the gall-bladder and the cystic duct but also the common bile-duct and common hepatic ducts were enormously distended with a similar clot, which probably extended into the ducts in the liver, so that no benefit could result from a further removal

of clot. No stone could be felt. She died the same night, being drowsy and apathetic, but capable of being roused into consciousness up to the last.

Dr. Clarke arranged for a post-mortem examination, and forwarded the parts to Dr. Lauriston Shaw, the curator of the museum at Guy's Hospital. The following represents his report of the conditions found, though it would be well to mention that the size of the gall-bladder and ducts with their contents diminished very considerably during the several days it was kept in spirit and water previous to dissection. "The substance of the liver, the duodenum and the colon appeared normal. The summit of the gall-bladder was sutured firmly to the abdominal wall, the skin of which was deeply jaundiced, and a large drainage-tube passed through an incision in the parietes into the gall-bladder. The gall-bladder was distended to about twice its normal size, and was completely filled with clotted blood. The cystic duct was dilated but not thickened. It had been cut in removing the preparation and did not contain blood. The common duct was greatly distended, and was completely filled with firm blood-clot, which extended into the main hepatic duct and into the branches of the ducts within the liver. Many of these ducts were traced throughout their course in the liver, and no diseased condition of their walls nor any communication with the blood-vessels was discovered.

On turning out the clot from the gall-bladder it was found that the mucous membrane lining it was lacerated in a longitudinal direction for a distance of about an inch and a half in the anterior wall, and that the rent extended for a small distance into the substance of the adherent liver. In the absence of any other discovered cause for the hæmorrhage, it appears probable that this laceration of the mucous membrane was its source, and that the blood having filled the gall-bladder made its way into the bile-ducts. The amount of distension of the gall-bladder found at the post-mortem examination will hardly warrant the view that laceration was the result of the distension, although its great size, as found at the operation, lends some weight to this suggestion. No gall-stone was found, nor any other cause for the jaundice than the obstruction of the ducts by the blood-clot. If the laceration was the cause and not the result of the hæmorrhage the only reasonable explanation of it would appear to be an injury."

As far as Mr. Clarke could learn from the friends, there

was no evidence of the patient having sustained any direct or external violence to account for the laceration of the mucous membrane of the gall-bladder and the liver.

On account of the very definite association of the sudden onset of pain in the position of the gall-bladder with the violent straining effort to expel fluid fæces, Mr. Clarke and I assumed that the rupture resulted from force exerted in this manner, and it would appear highly probable that such was the case. It is possible that the stone which produced the obstructive jaundice was forced into the bowel by the pressure of the blood behind it, which also exerted a very considerable dilating influence on the calibre of the already very much dilated common duct, and that it escaped unobserved in the evacuations which were thrown away by the friends.

In the *Transactions* of the American Surgical Association, 1893, I found in a short paper by Dr. Willard, entitled "Cholecystectomy for impacted gall-stones," the following report of a case, which, though unfortunately incomplete, would tend to some extent to support the explanation of the rupture given above.

"Hepatic colic; rupture of the gall-bladder; collapse; death. Male, æt. 40. For thirteen years he had had occasional attacks of sudden intense pain in the right hypochondriac region with vomiting and great prostration, such attacks being followed for a period of a few weeks by gastric irritation, after which he would be in good health for several months. He had an attack about one week previous to his death, but was relieved by chloroform, hypodermics, &c.

"He was at his usual duties as druggist during the day, but in the night was taken with a severe pain in the region of the gall-bladder. He was easier after a time and slept for an hour. About 6 A.M., when on the commode and straining, his wife noticed that he was in a cold perspiration. His voice became feeble, and when seen by Dr. Dick a few moments later he was in a state of profound collapse, pulseless at the wrist, cold, clammy, and unable to articulate. In this state I saw him in consultation at 10 A.M. practically moribund. Any operative interference was out of the question. The left side of the abdomen was flat over a large area of the left hypochondriac and left lumbar regions, probably from bile, blood, &c., probably from rupture of the gall-bladder or duct. He never had symptoms of aneurysm. The

abdomen was slightly tympanitic and distended, temperature 97°. Sinapisms, heat, hypodermics of nitro-glycerine, digitalin, strychnia, &c., slightly roused the heart, and he lived till 7 P.M. At no time could a radial pulse be detected. Autopsy refused. Evidently rupture of the gall-bladder, with hæmorrhage, a gall-stone having probably ulcerated through the duct." Though the diagnosis was not verified after death, it is very possible that the patient died of hæmorrhage consequent on rupture of a very vascular, subacutely inflamed and somewhat distended gall-bladder during the violent straining in much the same way as the urinary bladder will rupture under similar circumstances.

Such a sequence of events as occurred in my patient I have not read of, and I am bringing it before this Society to learn from the experience of others whether it is as rare as I suppose. It would appear that death resulted from the very complete obstruction brought about by the distension of the ducts by blood rather than from the amount of blood lost, which must, however, have been considerable and accessory in producing an early fatal termination.

XXVII.—*Traumatic Cystic Lympho-sarcoma of the Shoulder removed after five years' growth by amputation of the right upper extremity, the flap being taken from the upper half of the arm.* By Walter G. Spencer, M.S. *Read March* 8, 1895.

INTRODUCTORY.—The case here described is noteworthy both from the character of the tumour and from the operation required for its removal. The growth was of a rare structure, and whereas its early course was insidious, alarming symptoms attended its later career. The amputation just sufficed for complete removal, but it left the entire wound to be covered by a flap of skin from the upper half of the arm.

Patient's medical history.—J. M. C., aged 21, an accountant, is a native of New Zealand, where he has always lived, spending much of his time out of doors, and having had no previous illness. None of his family have suffered from malignant disease.

In April, 1889, the recoil of a gun bruised his right shoulder over and above the clavicle. Although the skin was not injured, yet afterwards pain was always felt whenever he shot off a gun. After some months a soft swelling was found to be forming at the seat of injury. In 1891 he took advice, the swelling was incised and "oily" fluid escaped. In February, 1892, chloroform was administered for the first time, a large opening was made into the swelling and the clavicle scraped; "oily" fluid, but never pus, was discharged.

From February to October, 1893, the swelling was opened several times and scraping done, after each of which operations the wounds healed with great rapidity. On October 1, 1893, the clavicle was removed (subperiosteally ?) and some cysts drained. In a month's time the swelling had become as big as before. In March, 1894, three openings were made and drainage-tubes inserted; the incisions still continued to close most rapidly, and had to be reopened every week. During this time he had several attacks of inflammation, and was once delirious. His right arm became swollen and his general health seems to have suffered, so that when he consulted three other medical men they said that nothing could be done, and that he might not live more than three weeks. He took ship for England forthwith.

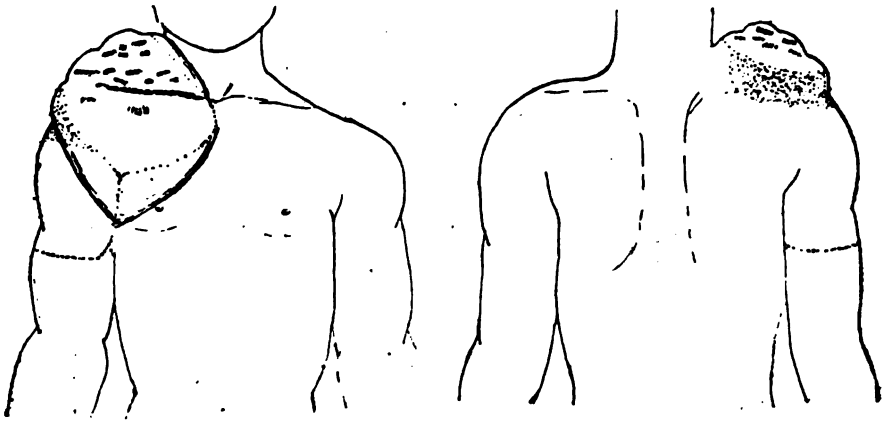
On the voyage all the drain-openings closed, the swelling of the arm disappeared, and he regained robust health.

On arrival in London he was taken by Dr. Frederick Mouat to see Mr. Macnamara, who being about to leave town kindly transferred the case to me.

The condition on examination.—The patient was found to be in perfectly good general health, stout and muscular, with a fresh colour, and thoroughly cheerful. No evidence of visceral disease nor any sign of secondary growths could be discovered.

Covering the right shoulder (see Figs. 12 and 13) was a

FIG. 12. FIG. 13.



tumour which extended from the third rib in the anterior nipple line to the spine of the scapula behind, and from the inner border of the sterno-mastoid muscle to the point of the shoulder. It stood up from the shoulder to the level of the angle of the jaw, and caused a bulging in the axilla below. On the dome of the tumour were numerous scars which marked the site of former incisions, and one long scar showed where the wound had been made for excision of the clavicle. The tumour was clearly cystic and multilocular, with solid material beneath the central portion, but none could be felt at the periphery. Over the prominent part around and among the scars the skin was adherent, but at the margin the skin was freely movable.

The tumour was fixed to the shoulder, but seemed to move a little with the latter over the deep structures of the neck.

The circulation in the arm was quite natural; there was no sign of compression of arteries, veins, or lymphatics. The only veins visible over the tumour were among the scars. The external jugular crossed the inner part of the tumour, having been pushed nearer to the middle line than usual.

Diagnosis.—On consultation with Dr. Mouat and Mr. Macnamara, it was agreed that the growth was probably an encapsuled cystic sarcoma, only locally malignant, seeing that after five years and in spite of long-continued irritation it had not involved the vessels. It was therefore proposed that the upper extremity should be removed provided that the encapsulation were confirmed upon incision, and provided that the vessels could be securely ligatured beforehand.

Operation, September 14, 1894.—Dr. Mouat and Mr. Macnamara were present, and I was greatly assisted during the operation and in the after-treatment by Messrs. Nowell, Dowler, Morton and Nelson.

Ether was administered and the usual precautions against shock were taken. I commenced by an incision (see Fig. 12) in a line between the mastoid process and the sternum from the level of the angle of the jaw to the junction between the second rib and sternum, and passing thence over the tumour terminated in the anterior fold of the axilla. After dividing some fibres of the pectoralis the bluish-grey capsule of the tumour was exposed, and it was found to easily shell out from the surrounding tissues. The wall of the cyst rent on forcible retraction, and material like broken-down brain escaped. This escape favoured retraction upwards, which would have been impossible in the original tense state of the tumour. The junction of the first rib with the sternum was the landmark made out, and on following its outer border backwards a rod of bone in the position of the clavicle about as thick as a lead pencil was reached, and a piece resected by bone forceps. Thus the subclavian vein was exposed and ligatured close to its junction with the external jugular. A second vein of the same size, perhaps an enlarged supra-scapular, also joined the angle between the external jugular and subclavian, and was similarly treated. Upon further retraction of the shoulder and of the overlapping cysts the outer border of the first rib was followed backwards to the scalene tubercle, and so the subclavian artery was found and ligatured immediately beyond the scalenus anticus to the proximal side of the origin of the posterior scapular artery. The rest of the pectoral muscle was then cut through, the cords of the brachial

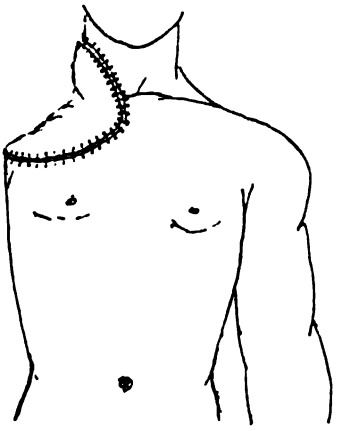
plexus divided, and the supra-scapular artery caught before division.

The posterior incision (see Fig. 13) was made by starting from the upper end of the anterior incision across the back of the tumour, along the upper border of the scapula, and over the point of shoulder, to meet the first incision again at the anterior axillary fold. The flap was then raised from the upper half of the arm by a vertical incision down the inner side, and a horizontal one round the middle, below the insertion of the deltoid, the fascia covering the muscles being included in the flap. When the patient had been rolled to his left and the right arm drawn well across the trunk, the trapezius, rhomboids, and latissimus dorsi were divided, only the posterior cervical artery spurting. The patient having been returned to the dorsal position and the arm drawn downwards and to the right, the tumour was shelled out from the inner side, where it was in contact with important structures, especially the internal jugular vein and pleura. The sterno-mastoid was smaller than normal, the clavicular portion having wasted after the removal of the clavicle. On separating the tumour the internal jugular vein was seen along the inner border of the wound, also the external jugular which had been retracted inwards. The dome of the pleura was blown up into the wound with respiration. The sterno-mastoid, the scalene muscles, the divided ends of the scapular muscles, and the upper two ribs, formed the floor of the wound. The omohyoid ran over the tumour, the levator anguli scapulæ was involved in the growth except at the upper part, where it was divided close to the transverse processes.

The skin of the upper half of the arm was not an excessive covering for the wound; the skin of the front and back of the neck being, of course, unyielding. This flap was a source of anxiety, for its blood-supply could only reach it from posterior cervical and dorsal vessels through the skin of the axilla and the posterior axillary fold. However, it lived well. The skin covering the spine of the scapula and the acromion, naturally thin, had to be raised with scarcely any subcutaneous tissue, and it formed a bloodless strip across which the circulation did not pass, and this determined the death of the skin beyond. To have avoided the sloughing which followed, the posterior incision should have included the area marked by dots on Figs. 12 and 13. The wound was sewn up by a continuous suture (see Fig. 14), and drains inserted

at the angles. The operation lasted 1½ hours. The patient soon became conscious, but remained clammy for twelve hours, after which there was a reaction during which he sweated freely; the pulse rose to 140, the respiration, which was

FIG. 14.



somewhat superficial and catchy, to 42, and the temperature to 101° F.

The wound was dressed for the first time four days after the operation; it was then seen that the patch of skin marked by the dotted area of Figs. 12 and 13 would ultimately slough as far as the line of skin which had covered the spine of the scapula and acromion. The dusky skin was raised and iodoform gauze slipped beneath it, and afterwards the wound was dressed twice a day, the sloughing skin being kept between two layers of gauze. It was impossible to avoid some suppuration, but it was mainly limited to the sloughing part, and all the line of union formed by the arm skin healed at once. A line of demarcation formed but slowly, so that the whole of the slough was not removed until the twenty-second day. During this time the divided muscles, which had been so well developed as to fill out the base of the flap for some days, gradually wasted, and then this base of the flap travelled forwards and upwards, compensating for the skin which had sloughed. Knots of silk from the ligatures on the vessels worked out of the upper part of the wound on the twenty-second day. During the fourth week he began to get up, and the ulcer left by the sloughing healed completely in eight

weeks (see Fig. 15). He quite recovered his former strength, and was provided with a shield to protect the side and to set out his clothes to the shape of the opposite shoulder. He left for New Zealand early in December; the appearance of the stump at that time is shown in the photograph (Fig. 15).

FIG. 15.

REMARKS.—*Pathological.*—The multilocular cystic tumour which I now show will be preserved in the Westminster Hospital Museum. It has much shrunken in size both from

the collapse of the cysts after the escape of fluid and from the action of spirit. It is firmly attached to the scapula, especially to the upper part of the venter, has involved the muscles, and surrounds the vessels beyond the point where they were divided. The intercystic solid material is small in amount, and has the appearance under the microscope of lymphatic gland tissue. The cells are similar to large lymph-corpuscles, consisting mainly of a nucleus with a very narrow zone of protoplasm around, and containing one, two, or three nucleoli. The delicate stroma between the cells is similar to retiform connective tissue.

Where this tissue lines the cysts, and is in course of breaking down, the cells have lost their logwood-staining property, and similar cells form the contents of the cysts.

If this tumour originated from normal tissue of the same kind, it must have grown from the subclavian lymph glands; and if this was indeed the case, the course of the omohyoid across the tumour would be explained. When the patient says that "oily" fluid came from the cysts in the earlier stages, I understand him to mean serous fluid, as only a dermoid cyst, which this tumour is certainly not, yields oil.

My difficulty in calling the growth lympho-sarcoma is twofold, viz. the traumatic origin and early cystic character. Hygroma is the name given to congenital multilocular cysts; they are not traumatic in origin, have fibrous tissue, not lymphatic gland tissue, between the cysts, and a serous lining.

Clinical.—The first incision assured me that the growth could be easily separated from surrounding parts. This encapsulation was an all-important factor in isolating the vessels and in separating the tumour from the internal jugular vein and from the pleura. By ligaturing the vein first, the limb was removed full of blood; but this is in accordance with present practice, and except by oozing no more arterial blood was lost. The subclavian vessels could not in this case have been reached from above; but of course the method of getting to them from below is only applicable to such a case as this, for the vein has to be divided first before the artery can be exposed. The method allowed me to take the first rib as a sure guide to both the vein and artery. The new bone which was divided had formed, I suppose, from periosteum left behind in the removal of the clavicle. The skin which now covers the site of the wound came entirely from the upper arm, and was from the first well supplied by blood.

The result of the sloughing was to remove the skin included by the dotted area on Figs. 12 and 13. I recognised at the time how thin was the skin raised from the acromion and spine of the scapula, but I had no precedent to assure me that such an arm flap would live upon the blood-supply from posterior cervical and dorsal vessels, and I therefore kept all the skin I could.

Retrospective.—The case was clearly a very difficult one from the first. The slow development of the cysts naturally suggested the treatment by drainage, whilst later on a diagnosis of myeloid sarcoma growing from the clavicle after an injury was naturally formed on the score of probability. As the tumour continued to grow, its complete removal could only have been accomplished by an operation attended by great risk to the arm. Granted, indeed, that the inevitably wide loss of skin could have been replaced by transplantation, would not an attempt to remove the whole of the tumour have led to ligature of the subclavian and suprascapular veins and so to gangrene of the arm, for when the growth began to exhibit malignant characters it was already tending towards the axilla. Therefore I cannot see how any operation short of the one performed by me could have cured the patient even at a much earlier stage.

The exhaustion from the treatment by drainage, together with the septic complications which had caused the arm to swell, seemed to those who saw him just before he left New Zealand to indicate an early fatal termination from secondary growths. On the other hand, the sea voyage, by restoring his general health and by eliminating the septic complications, showed that the disease was still circumscribed, and prepared him in a most satisfactory manner for a severe operation.

Prospective.—He has now returned to New Zealand. The slow development of the lympho-sarcoma during five years, its encapsulation, its complete removal, and the absence of all evidences of metastasis, enable one to give a good prognosis.

XXVIII.—*A case of Complete Unilateral Arrest of Development, with arcus senilis, and without hemiplegia, in a girl aged twelve years.* By William Wallis Ord, M.D. *Read March* 8, 1895.

S. M., æt. 12, has been under my observation at the Victoria Hospital for Children for some years. She is the third of six children. The second child, a boy, died of hydrocephalus while the patient was *in utero*. The mother nursed him in his illness, and was greatly distressed thereby. The other children are healthy, and there have been no miscarriages. There is no history of epilepsy, insanity, or any form of nervous disease either on the father's or mother's side.

The patient was born at full term; delivery was natural, and no instruments were used. Her mother states that she noticed from the first that the arm and leg on the right side were smaller than those on the left. There was, however, apparently no paralysis nor any rigidity. The child began to walk at about the age of eighteen months, but was always meeting with severe falls on account of the weakness of the right leg. Till the age of four years she was always considered to be "backward," but no history can be obtained of any delay in dentition. She began to speak at about the age of two years; her speech has never been defective. She has never had any fits, and with the exception of measles and two attacks of influenza has enjoyed fair health. She has always suffered from attacks of headache, which have of late increased considerably in severity.

The patient is a thin, delicate-looking girl, who walks with a slight limp, and with the left shoulder raised. Her facial appearance is striking. Below the level of the eyebrows the right side of the face is markedly smaller than the left. Above this level the proportions are reversed, the left side of the cranial vault being smaller than the right. The differences on the two sides affect alike bony and soft structures. With regard to the facial muscles, those on the right side, although the smaller, seem to act more strongly than those on the left. This I consider to be only an apparent difference, due to the fact that there is less fat to mask their movements on this side. At all events there is

no loss of power in the muscles on the right side, as may easily be seen by putting the masseters in action.

Fig. 16.

Photograph of S. M.

The palpebral fissures are equal on the two sides. The slight difference apparent in the appearance of the globe of

the right side, as compared with that on the left, is attributed by Mr. Holmes Spicer (to whom I am indebted for the following particulars with regard to the eyes) to a deficiency in the orbital fat, and to a want of development of the

FIG. 17.

Photograph of S. M.

margin of the orbit. The pupils are equal and act to light. There is no paralysis nor weakness of any of the ocular muscles, and there is no defect in accommodation, which is equal on the two sides. The movements of the eyes are

perfectly free in every direction. There is nothing abnormal in the fundi, and there is no hemianopsia. In the right eye there is a thin, sharply-defined arcus above and below. The left eye is emmetropic, V. = $\frac{6}{6}$. In the right there is considerable myopic astigmatism, with −2 D. Cy., V. = $\frac{6}{12}$. There is no difference in the amount of hair on the two sides of the scalp, or of the eyebrows or eyelashes.

The ears are equal in size.

The tongue is protruded straight, and its movements are good. To the eye the right side is evidently smaller than the left, and to the touch the right side is distinctly more flabby than the left. Faradic sensibility unimpaired. Smell, taste, and hearing are natural.

On stripping the child a similar difference between the size of the limbs and trunk on the two sides is observable. The following measurements will demonstrate approximately the difference:

	Right.	Left.
Clavicle	3¾ inches.	4 inches.
Arm, from acromion to tip of middle finger	18	19½
Girth of arm	5¼	5¾
,, forearm	5½	6
Length from ant. sup. spine to tip of internal malleolus	23	24¾
Girth of thigh	11¾	12
,, calf	8½	9
,, chest at level of the nipples	10¼	11½
Girth of head, mid-occipital to mid-frontal	10	9½

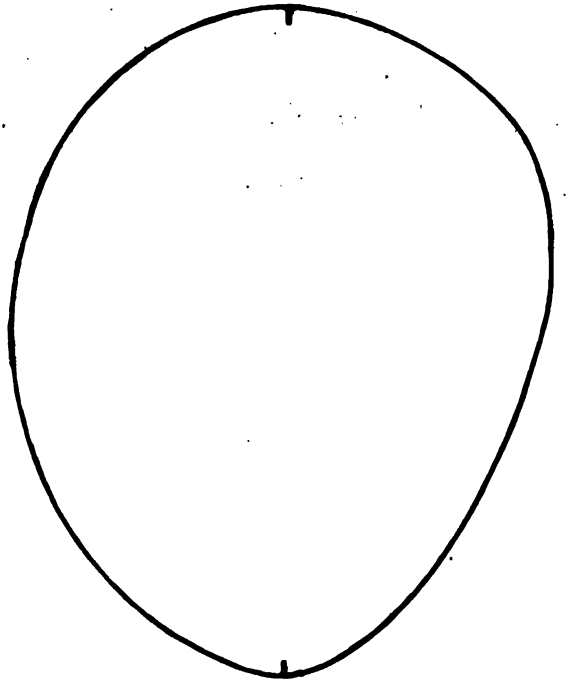
There is nowhere any loss of sensation. There is no paralysis, but the muscular power on the right side is everywhere less than that on the left, *e. g.* the hand grasp.

All voluntary movements are free, there is no trace of contracture, and no inco-ordination. Faradic sensibility is normal on the two sides. The superficial reflexes are present. The patellar reflexes are absent on both sides, and there is no clonus. The child's intellect is good, but she is rather emotional. She is said to be clever at her lessons.

In this case we have a deficient development of the whole of the right side of the body below the level of the

eyebrow, involving bones as well as soft structures. Above the level of the eyebrow the conditions are reversed, and the left side is the smaller. The pathology of the case is obscure. Obviously it has relations with the condition known as infantile spastic hemiplegia, but differs from that condition in there being no real paralysis, no rigidity, and no

FIG. 18.

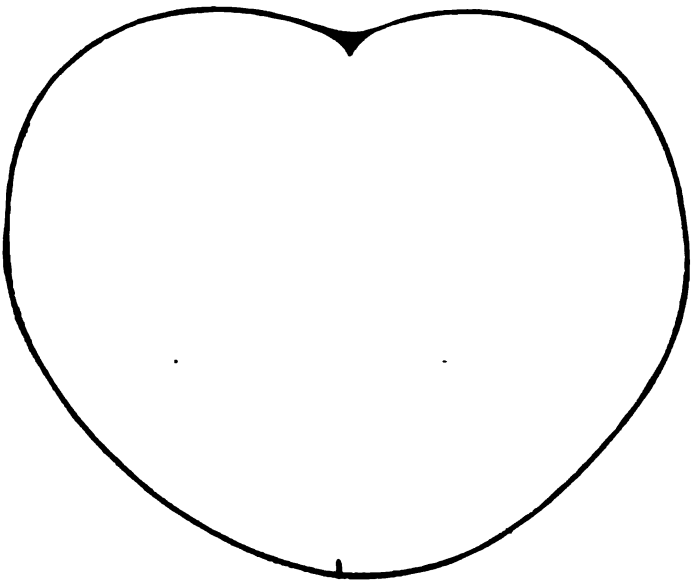


Cyrtometric tracing of head just above level of eyebrows.

mental deficiency. The comparatively small size of the left side of the skull would seem to point to an intracranial lesion on the left side of the brain, which has led to contraction, or a loss of substance on that side. Is the case an error of development, or is it due to a destructive lesion? The mildness of the case, and its non-progressive character, may seem to point to the former. With regard to the arcus I have no note of its presence before October, 1894, but I had not seen the child then for some months. I am strongly under the impression that this phenomenon has only

appeared recently. If this is so, perhaps some ophthalmic authority could say if this would throw any light on the pathology of the case. In this regard I may mention two cases published by Mr. Jessop and Dr. Oswald Browne in vol. xviii of *St. Bartholomew's Hospital Reports*. These cases were described under the heading of "hemiatropia facialis." They do not appear to have closely resembled this case, except in one point, that there was progressive atrophic trouble in the eye of the affected side in these cases, viz. "narrowing of the palpebral fissure, sinking in of the eye, atrophy of the orbit, dilated or contracted pupil, corneal nebulæ and ulcers, conjunctivitis, lagophthalmos, and later, as a result of these trophic changes, destruction of the eye."

Fig. 19.



Cyrtometric tracing of chest at level of the nipple.

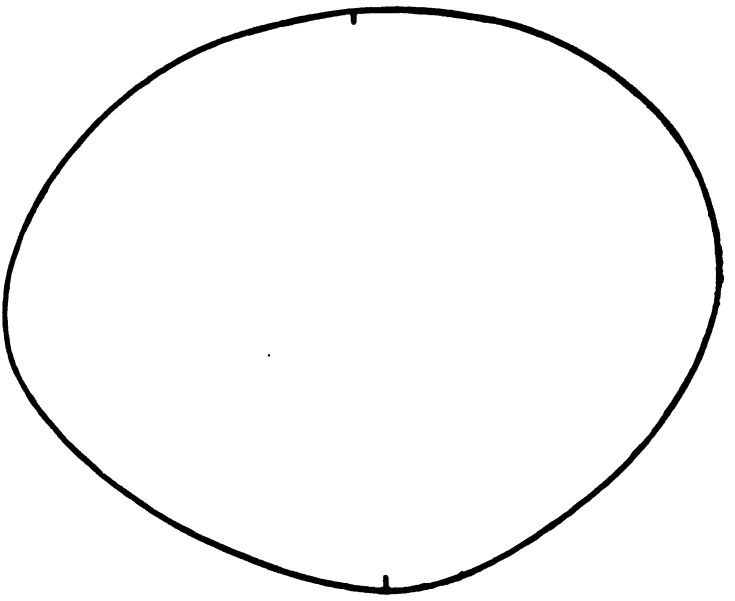
Whether the corneal ulceration in these cases, and the arcus in the one before you, may show any connection between the cases is an interesting question.

In 1887 Dr. Judson S. Bury, of Manchester, presented a case, very similar in most respects to mine, before the Medical Society of Manchester, and he has kindly lent me

his notes, an abstract of which appeared in the *British Medical Journal* of that year, vol. i, p. 458. There were no eye symptoms in his case, but the difference in size of the two sides of the tongue was very much more marked than in this. A figure of his case appears in his *Clinical Medicine*. There was a history of "fits" in the mother during pregnancy, corresponding to the mental strain and anxiety in the same period in this case. Otherwise the two cases appear to be identical, and I may with advantage quote his remarks on his case.

"Whether the porencephalic defect is due to defective

FIG. 20.



Cyrtometric tracing of pelvis.

development or to a destructive lesion is not very easy to say; the fœtus may have suffered from convulsions when the mother did, and it is conceivable that the fits disturbed the cortex, and led to impairment of the function of the ganglionic cells there, and so to impaired development of the motor area on that side."

On the whole I am disposed to regard it as an extremely mild form of infantile spastic hemiplegia.

XXIX.—*Case of Acute Intestinal Obstruction due to volvulus of the sigmoid flexure: abdominal section: recovery.* By F. Lucas Benham, M.D., and A. Quarry Silcock. *Read April* 26, 1895.

MRS. X., æt. 56, widow. Had lived in India for the first thirty years of her life; while there, about thirty years ago, she had a sunstroke, in consequence of which her mind became affected, and she has remained permanently in a state of chronic mania. She is harmless, gentle, and tractable, seldom getting much excited; but is almost constantly talking to herself, holding a rather incoherent dialogue between herself and imaginary people. She is observant of what passes, but cannot attend rationally to anything. She is generally clean in her habits, and is not mischievous or destructive. Some time ago she was confined for a while in an asylum; but being harmless and not requiring to be kept under lock and key, she was removed and has since been under private care in the country. Some months ago she had some dropsy, which has quite passed away. On Thursday, November 1, 1894, she came up to town with the lady who takes charge of her. She seemed quite well at the time, though on the morning of her journey her abdomen was noticed to be somewhat fuller than usual. She had slipped while walking in the garden a few days before, but this did not appear in any way serious and but little notice was taken of it. She has always suffered from obstinate constipation, requiring frequent aperients to overcome it; but for several days before coming to London the bowels had acted with unusual regularity, more or less daily.

On November 2 the bowels were confined and she seemed uncomfortable. A dose of Cascara Sagrada was given without effect.

November 3.—She was kept in bed in consequence of having taken medicine overnight, and an enema was given without any result. In the evening she had ½ oz. castor oil, which made her sick, and some fish which she had had for dinner was also vomited in the night.

November 4 (Sunday).—Patient seemed ill and vomited

frequently, whatever was taken. She also vomited about 2 oz. of black stuff like coffee-grounds (altered blood?) Bowels not open. Seemed to have some pain in abdomen, chiefly on the left side.

I was sent for and saw her about midday. She was then lying in bed on her left side, with her legs somewhat drawn up, and looked thin, weak and anxious, face dingy and pinched. Was very quiet; seemed demented; did not speak except for talking to herself in a low voice; understood and would respond partially—*e.g.* would protrude her tongue when asked to. Tongue clean and moist. Pulse 84, small, regular. Abdomen much distended, moderately tense; no tenderness; no fluctuation; tympanitic all over; colon evidently occupying whole of flanks, and reaching high beneath ribs; peristalsis not visible. Rectum empty; no fæces; no tumour or any other morbid condition felt.

Diagnosis.—Intestinal obstruction, probably in the sigmoid flexure or lower part of descending colon. Cause doubtful.

I gave an enema of castor oil ℥j, olive oil ℥viij, and plenty of soap and water. Most of this returned at once without any fæces. I then ordered calomel gr. iij with rhubarb gr. v, which was vomited almost directly.

At 4 P.M. she seemed a little better, but had vomited milk and soda water which she had swallowed, though she had kept down a little milk and whiskey. Bowels not open. Slept frequently. I thought it desirable that a surgeon should see her with a view to a speedy operation; so at 7 P.M. I had a consultation with Mr. Silcock. There was then a very small quantity of fæces in the rectum, and we agreed to have one or two similar enemata given during the night, and to operate next day if there was no relief. Temperature 99·6°.

November 5.—Two enemata had been given without any effect, and caused some pain. Patient had been continually sick; had kept down a little whiskey but nothing else, and had vomited some more of the coffee-grounds stuff, but there was no stercoraceous vomiting. Patient looked weaker; pulse 96.

At 3 P.M., after two enemata of brandy and beef tea and 1 oz. brandy by mouth had been given, I put her under chloroform, and Mr. Silcock, assisted by Mr. Crowle, performed abdominal section. During the operation, which lasted about 1¼ hours, she was very slightly sick.

Notes of the operation (by Mr. Silcock).—The abdomen

was opened by a four-inch incision in the middle line below the umbilicus. Beneath the wound lay an enormously distended piece of large intestine forming part of a loop which reached upwards to the diaphragm, and below to the brim of the pelvis. At this level and to the left of the middle line a twist of the gut could be felt, and the lesion was at once recognised as that of volvulus of the sigmoid flexure. The involved loop of intestine was congested, but not extremely so. It was so distended that it could not be delivered through the wound, therefore it was punctured with the needle of a hypodermic syringe in order to let off the contained gas. The latter escaped very slowly, hence a larger vent was made by passing a tenotomy knife through the walls of the gut transversely to its long axis; through this opening a large quantity of gas and some fluid fæces were evacuated. Every precaution was taken to prevent the fæcal matter from infecting the peritoneum during this procedure. The distension of the bowel being thus materially lessened, the twisted loop could now be easily brought out through the wound in the parietes. As much fæcal matter as would readily flow through the small opening was allowed to escape, then the wound was closed by means of three Lembert's sutures, fine silk being used. The twist was undone by turning the right-hand section of the loop forwards and to the left over the left-hand section. (From this it will be obvious that the volvulus was the result of twist of the sigmoid flexure through half a circle in a direction forwards and to the right).

As soon as the volvulus was reduced and the gut restored to its normal position, the contents of the distended loop passed onwards into the rectum. In order to facilitate the expulsion of the retained fæces, the vaginal tube of a Higginson's syringe was passed through the anus, and through this large quantities escaped, aided by gentle manipulation of the distended and paralysed gut.

The peritoneum was cleansed in the usual manner, the parietal wound closed and dressed with cyanide of zinc and mercury dressings, a firm flannel bandage being applied over them.

The wound was dressed on November 8th, 12th (three stitches removed), and 16th (rest of stitches removed), and was quite healed on the 20th.

After the operation was over she was very much easier, and did not vomit at all. Pulse 84, slightly uneven; tongue clean and moist; temperature 100·2°.

After the operation the patient did very well, and gradually returned to her natural state, without any complication of any consequence.

The course of events may be epitomized as follows:

She had no abdominal distension or pain whatever, but throughout convalescence lay quietly in bed in a passive condition, seeming to take but little heed of what had taken place, sleeping by night, and talking to herself in a low voice or out loud most of the day.

The bowels acted very freely. A large quantity of pulpy grey fæces passed directly after the volvulus had been reduced, and continued to do so at short intervals for the next two or three days, and then the stools were more lumpy and slimy.

A small dose of castor oil was given on November 11th and again on the 13th, which had good effect. From the 15th onwards the bowels were confined, and were relieved speedily and satisfactorily by an enema on alternate days.

There was no vomiting at all.

The tongue was moist directly after the operation; but next day it became very dry and brownish, and kept more or less dry for some time, not becoming altogether moist and natural for more than a fortnight. I am inclined to think that this was due to absorption of iodoform, for the dryness diminished as the raw surface healed, and quite disappeared directly the iodoform was no longer required or used, and there was no other cause that I am aware of to account for this dryness.

The temperature was a little over 99° before the operation, and rose to 100·2° in the following evening. From this time it never rose so high, but varied generally from 98° to 99°, occasionally falling below 98°, for nearly a fortnight, and after this was normal or subnormal.

The pulse was 96 before the operation, 84 immediately after. For two or three days it was slightly intermittent. Its rate remained a little over 80 till the temperature became normal, and then dropped gradually to 70 and 66.

Respiration normal throughout. Diaphragm descended freely. There was no cough. Urine scanty, dark and turbid for two or three days, and then became abundant and clear. Some care had to be taken to keep the patient clean and dry, as, owing to her mental condition, she would often, if not always, pass urine involuntarily, or without giving the nurse any notice.

Diet.—Patient was given nutrient enemata only for

thirty hours after the operation; but the bowels were acting so frequently that this method was not at all successful. Food was given by the mouth on the second night following the operation in the shape of 2 oz. arrowroot and milk or 2 oz. beef tea, each with ½ oz. brandy, at intervals of four hours. On November 7 she was ordered one pint each of milk (with Liq. Calcis Sacch.), barley-water, and beef tea, and 4 oz. brandy, in twenty-four hours, in small quantities at short intervals. This diet was by degrees changed for more abundant, varied, and solid food. She took all that was given her readily, and without suffering any ill effects. As she had no teeth, regard had to be paid to giving food always in a condition that did not require efforts of mastication.

She was kept lying on her back for twenty-one days, and during all this time she remained perfectly quiet and did not meddle with the dressings. In a few days after being allowed to sit up she was dressed and able to walk about, looking very little the worse for her accident.

Remarks.—We considered this case worthy of being brought before the Society because of the rarity of cases of volvulus that have been cured by operation. In fact we have been able to discover only one such instance on record of recovery after abdominal section. This other case is narrated in the *Lancet*, 1883, vol. ii, and was under the care of Mr. Clarke, of Glasgow. It was in some respects remarkable, the obstruction having lasted about three weeks before operation seemed imperative, and was unattended throughout by vomiting.

Ours, on the other hand, was more acute.

I think it was impossible to determine before opening the abdomen the nature of the obstruction; possibly the absence of visible peristalsis might have led one to suspect that the colon was displaced, possibly from a twist. On the other hand, the coffee-grounds vomit made one take into account the possibility of there being some cancerous tumour involving the stomach; but I thought it most likely that the hæmorrhage was merely due to congestion of the mucous membrane of the stomach and straining with vomiting, and did not attach much importance to it.

Also the leanness and Indian complexion of the patient went further to suggest cachexia from the presence of malignant disease.

I will not enlarge upon the pathology of the disease, as, though not much is precisely known about it, it is probably a

simple mechanical affair. I believe, however, in this case that the bowel got somewhat displaced and twisted by the slip she had met with, as mentioned above, while it was loaded with a large accumulation of fæces; that the slight twist could not rectify itself, but became aggravated by the natural peristaltic movements.

It is well known that in some insane patients the colon is misplaced and contorted. I have myself seen two or three times at least the peculiar V-shaped bend of the transverse colon towards the pubes; but I do not see how to ascribe the affection to such a condition, and volvulus or obstruction is not, I think, especially common among the inmates of asylums.

Postscript.—Since writing this paper, it has come to our knowledge that other successful cases, eight in all, have been reported in addition to and later than the only other one quoted above. All were on the Continent. The following are the references to them:

1. Braun (*Archiv für klin. Chirurg.*, vol. xliii, p. 164).
2. Senn (*Medical News*, 1889).
3. Lindshedt } Recorded in Swedish journals, and quoted
4. Liden, } by Braun in the article mentioned
5. Ussing, } above.
6. Obalinski (*Wiener med. Presse*, 1884, No. 48; 1885, No. 8). The operation was successful, but the volvulus returned in some weeks, and the patient died.
7. Kiwall (*Petersburg. med. Wochenschrift*, 1893, No. 49).
8. Onlowski (*Chirurg. Centralblatt*, 1888, No. 48). But in the report of this case it is not distinctly stated whether the volvulus was of the sigmoid flexure or of the small intestine.

Besides these successful cases there have been some others reported which have been partially successful, *i. e.* where, after the operation was immediately successful, the condition has soon returned and caused death, or the patient has died of some complication before he actually recovered, or else he has recovered with an artificial anus.

XXX.—*On Early Excision of the Sacro-iliac Joint, with cases.* By C. H. Golding-Bird. *Read April 26, 1895.*

THE very unsatisfactory prognosis that all surgeons agree must be given in tubercular sacro-iliac disease when once disorganisation of the joint, as indicated by the appearance of suppuration, has become evident, must be my reason for bringing the subject of the operative treatment of this affection before this Society; and I hope to show that by thorough and early excision (for it is more than mere "erasion") results quite as favorable may be obtained, as in the case of joints which from their anatomical relations are more easy of access.

I need not back the disconsolate statement I first made in any other way than by an appeal to individual surgical experience, and to the very admirable and exhaustive summary of our position in regard to inflammatory disease of the sacro-iliac joint presented to this Society last session by Mr. Makins; yet it is somewhat encouraging to read toward the end of his paper that in his opinion "the prognosis is better than is generally stated," and this opinion he bears out by quoting three cases in which he had undertaken operation in the suppuration stage, and in two out of the three (at the ages of three and twenty-five years) good results followed, whilst in the third, aged three and a half, the result was at least satisfactory as far as it went.

In April, 1880, and again in 1882, in the persons of two men, aged twenty-seven and twenty-six, I had the opportunity of operating when the disease was far advanced and the joints disorganised. I reached the joint in each case by following up sinuses, and in each removed sequestra. One case was discharged in due course, cured all but a sinus; but the other succumbed to blood-poisoning. In 1880 there was also under my care a lad with advanced sacro-iliac disease, but in this case, instead of following up sinuses I trephined the iliac surface of the joint, and thus was enabled to clear out sequestra through the opening, and give for the time complete relief to all symptoms. A year later he again came under my care with a return of his symptoms, and again I opened the joint in the same way; he was discharged in due course well, but with

a sinus. I heard of him as about at his duties a long time afterwards, but eventually lost sight of him.

This is the case referred to by Erichsen as a successful one of trephining the sacro-iliac joint; and I only refer to it now as emphasising Mr. Makins' more promising prognosis just quoted, and as being an early departure from the more common practice of following up sinuses to the joint, by deliberately opening it at its most superficial part.

In the *Lancet* for October, 1889, Mr. Mayo Collier described in detail a case in which he also trephined, with a most satisfactory result, the patient being cured; but the special interest in his case is the early stage in which he operated. No sinus had formed, and apparently no suppuration, though over the joint was a swelling the size of the palm of the hand. In his concluding remarks he urges operation where suppuration is threatening, or even where pain is severe and persistent. This opinion I cordially endorse, but I would even go further and urge that where even the pain is moderate, yet persistent and recurrent—yielding, it may be, to rest, but as surely recommencing when activity is resumed—we should act as we would in all other cases of tubercular arthritis, and thoroughly remove the disease in its earlier stage. This opinion, too, is supported by the excellent results I obtained in the cases I am about to mention.

I should, however, here remark that whilst my experience of sacro-iliac disease is all but entirely drawn from tubercular affections of the joint, yet the various pyæmic affections of it may sometimes offer an opportunity for radical operation, though I fear not often. Even the tubercular inflammation of the joint by no means offers in every case an equal prospect of recovery after operation; and the prognosis depends, I believe, not more on the degree to which the disease has reached, than it does on the place of origin of the affection.

When the disease begins in the sacrum the case is probably less hopeful than when, if it ever does so, it begins in the ilium; whilst it will be well imagined—and my cases seem to prove it—that the best results are likely to follow where the disease commences in, and is confined to, the articular surfaces themselves; in a word, a sacro-iliac primary arthritis is more favorable than an arthritis following epiphysitis. Perhaps I should not use this word, since there is but one epiphysis—the sacral—entering into the structure of the joint; yet this anatomical fact emphasises the doubt I

have just expressed as to whether tubercular arthritis of the sacro-iliac joint ever begins as an osteitis of the ilium.*

There is one point in the early recognition of these cases deserving of attention. On reading the clinical history of cases of sacro-iliac disease it is noticeable that nearly every patient has first been treated for sciatica, and if a woman, as likely as not for some supposed ovarian or uterine origin for her pain. At this early stage the time-honoured method of testing the effect of pressure exerted on the anterior superior iliac spines is likely to give negative results, and even pressure over the posterior sacro-iliac ligaments may not reveal anything; but experience has led me to think—and one of my cases was a marked instance of this—that in quite an early arthritis direct pressure over the joint itself will elicit pain. Pressure over the posterior ligaments is too far removed from the articulation; but when exerted directly on the ilium, at the root of the posterior inferior iliac spine, the bony surfaces of the joint are jammed together and tenderness is at once complained of, and the origin of the sciatica or other subjective symptom is made evident.

Since the operation performed was the same in all my cases, a general description of it first will avoid repetition.

The patient lying on the opposite side, a semicircular flap of skin and subcutaneous tissue over the iliac area of the joint, and having its convexity corresponding to the posterior edge of the ilium, is dissected upwards and forwards, and the underlying glutei are detached. The bone being thus freely exposed, a large trephine is applied at the root of the posterior inferior iliac spine, and in a line drawn from the top of that spine to the junction of the anterior with the middle third of the iliac crest. This line lies in the long axis of the auricular surface of the joint; and from measurements that I have made, it seems to me to be a safer guide than a line drawn to join the anterior superior spine of the ilium.

The ilium at the seat of operation is very thick, but the disc of bone removed should reach quite down to the joint. This opening enables an inspection of the articulation to be made, if that is all that is desired; but to remove disease it must be further enlarged by whatever means the surgeon thinks best, but it is essential that the amount of bone removed should be practically that forming the iliac surface of the joint.

* Lannelongue (*Tuberculose vertébrale*, p. 331) states that sacro-iliac disease always begins as an articular osteitis.

The diseased structures are now dealt with as in any other tubercular joint, frequent flushings with aseptic liquids being employed. When all visible disease has been removed, and especially if the trephine openings do not quite cover the area of the joint, the ilium should be prized off the sacrum with an elevator, and sufficient separation between the two bones is thus easily given to allow of the introduction between them of a Volkmann's spoon. A further gain by this manœuvre is that it may be the means of discovering pus lying just in the pelvis but outside the joint, and not before suspected. This was well shown in one of my cases.

Sinuses present will of course be treated according to general surgical rules; but I would express my opinion that the following up a sinus, as an indication of the route by which to reach the joint, is useless. The excision must be deliberately carried out on anatomical lines, and then the sinuses dealt with.

The superficial structures are now replaced and united by continuous suture, without any drain being employed, but after the introduction of iodoform emulsion; and though I have not employed a drain myself it is evident that the state of the parts after operation, as well as the practice of the operator, must really determine this question.

It seems at first sight as though such an extensive removal of bone must weaken the part and delay the patient's moving for a long time. In one case I did employ a Thomas's splint for many weeks; but in another, an adult male, I gave no support at all except that of a belt and of a crutch for a few days, and from the first of his putting his feet to the ground eight weeks after operation there was no suggestion of want of supporting power. The fact is that the posterior sacro-iliac ligaments prevent any gliding of the ilium on the sacrum, and that the joint, as such, takes little share in support. In none of the cases was any splint used immediately after the operation, movement of the leg and turning about in bed being encouraged.

Case 1.—Louisa W., æt. 14, was under my care in Guy's from May 21 to July 28, 1894. A year previously she had been seized with severe pain in the right sacro-iliac region; there was no history of injury. For the year she limped about at her work, not even using crutches, and the pain at last drove her to seek relief. When examined she gave all the usual subjective symptoms of sacro-iliac disease: indirect

pressure on the anterior iliac spines gave pain, and especially did she complain on pressure being made at the root of the posterior inferior iliac spine. There were no local objective symptoms whatever.

I decided to explore the joint, and performed the operation already described. On removal of the disc of bone by the trephine, a thick washleathery layer of lymph was seen; it covered the entire joint, and on removal of sufficient bone it was easily taken away. Granulation tissue and *débris* of cartilage were also removed; but no disease or softening of the bone, either iliac or sacral, could be determined. The operation completed, the wound was closed without a drain, and primary union resulted. She was sent out in eight weeks on crutches, wearing a Thomas's splint. This splint she wore for six months, but for the reasons already given I think this was an excess of caution. When last seen in January of that year she was perfectly well, and going about as usual.

Case 2.—John H., æt. 29, having suffered some time from sciatica, came to the medical department of the out-patient's at Guy's in May, 1894, for treatment. Being regarded as a case of sciatica, galvanism was ordered and some relief obtained. Four months later, as there was no material improvement, he was admitted into a medical ward and further treated; but a surgical origin for the pain being suspected, he was transferred to a surgeon's care, and the sacro-iliac disease being now suspected, he was encased in a plaster-of-Paris spica and sent home. Three months later, January 17, 1895, he was admitted into my ward.

On examination all the subjective symptoms of right sacro-iliac disease were present—pain was elicited by both direct and indirect pressure, but the worst symptom of all was the constant and very severe sciatica. There were no local objective signs at all.

On January 25 I operated after the method described, and in the joint, which was disorganised, were seen sequestra of cartilage and bone and pulpy granulation tissue, whilst the sacral aspect of it in parts was superficially soft, scraping away easily with a steel spoon. No pus was found in the joint cavity; but on prizing the bones asunder, pus welled into the joint, to the extent of only a few drachms, from two positions—one anteriorly from a subperitoneal cavity close by the lumbo-sacral cord, the other posteriorly at the exit of the great sciatic nerve. In order the better to deal with

this collection, some fibres of the great sacro-sciatic ligament were divided; both abscess cavities were thoroughly scraped. The wound was eventually closed without drainage, and primary union resulted with a complete subsidence of all the previous symptoms.

At the end of the fifth week the patient was allowed to get on to the couch; and at the end of the seventh week began to stand with the help of crutches, but these he discarded in a few days for sticks, and these again he ceased to use, and was able to walk and sit about the ward at the end of the ninth week: he is now (July, 1895) perfectly well.

Throughout convalescence no mechanical support was given except such as a firm pelvic belt afforded.

Case 3.—Miss B., æt. 30. In May, 1893, she fell on her back in a carriage accident; she was not much hurt, and little was thought of it until, at the end of two months, a pain in the lower part of the back, which had resulted from the accident, was found to be persistent.

I saw the lady, it being believed she had some spinal affection coming on. I could find none; and the pain being unilateral and not central, I employed the usual tests to discover sacro-iliac disease, without result, and there were no objective symptoms. However, on firmly pressing on the root of the posterior inferior iliac spine of the affected side, tenderness was complained of.

The diagnosis made was arthritis of the right sacro-iliac joint, due to the accident, and its persistence, together with the history and appearance of the patient, made one suggest the possibility of tubercular infection.

She for a time improved under care, but soon disregarded directions, and when I next saw her, in March, 1895, I found she had been months in bed, and the local signs were as before, only aggravated; there was still nothing objective.

I proposed and shortly after performed the operation described, and found a joint with many adhesions, as of repair, and evidence of disorganisation in sequestra of cartilage. I saw no proof of tubercular disease, though it might have been such.

Primary union followed, and a report sent me on April 8 was that by the end of the fortnight from the operation the patient was on the couch, primary union having resulted and an entire absence of all joint symptoms from the day of the operation. A still later report received from her medical

attendant, Mr. Lockhart Stephens, of Emsworth, is to the effect that all sacro-iliac symptoms have vanished, though there is some reason to suspect a focus of tubercular disease in the dorso-lumbar area of the spine.

By way of summarising my remarks I would urge these points:

The value of direct pressure over the most superficial part of the joint in discovering inflammation of it in an early stage.

The importance of operating as soon as this early condition has been established; and although under the best hygienic circumstances recovery, on expectant treatment, might result, yet operation offers relief in fewer weeks than mere rest does in months.

Operation on the joint should be conducted on anatomical lines, and not be carried out along the route of sinuses; these never open at the point where the joint is most superficial.

The steps of the operation should be calculated deliberately to expose the joint cavity and allow of its perfect cleansing, as well as of removal of even outlying matter; and not be conducted on the lines of a mere sequestrotomy, even in an advanced case.

Mechanical support does not appear necessary during healing, and afterwards a pelvic belt does all that is required with the aid of a crutch or stick.

Even in an adult weight may be borne by the part, in the erect position, at the seventh or eighth week—supposing, that is, that primary, or at least very early union of the parts operated upon is obtained.

XXXI.—*Two cases in which Orchotomy was performed for Enlargement of the Prostate.* By C. Mansell Moullin. *Read April* 26, 1895.

THE first case was that of a patient 74 years of age, who was sent to me by Dr. Birch, of Lower Clapton, suffering from advanced prostatic disease. The patient was a slightly-built man, very thin, and giving the impression of being utterly worn out by pain and loss of sleep. *Per rectum* the prostate was of enormous size. The history showed that it had been enlarged, and that the patient had been accustomed to use a catheter, for many years. The urine, in spite of the bladder being regularly washed out, was exceedingly foul, ammoniacal, and loaded with mucus and phosphates. The bladder was so contracted from years of cystitis that it could only hold two ounces of fluid, and there was the most intense strangury. The patient had to wear a portable urinal during the daytime, and in spite of morphia suppositories was compelled to pass his urine fourteen or fifteen times at night. The question of orchotomy was discussed with him, care being taken to point out that though the obstruction would be removed, it was not probable that the condition of the bladder could be much improved, but the misery was so great that the patient agreed at once. The operation was performed on October 26. There was no shock, and, though one of the ligatures slipped and caused considerable hæmorrhage, no after trouble with the wounds. Three days later it was noted that he had been able to dispense with morphia for two nights, and that he had slept for an hour and a half at a time, the longest spell previously having been three quarters of an hour. Ten days later the patient was examined again. The prostate was decidedly smaller as felt *per rectum;* the stream of urine was larger: the urine was neutral in reaction, and small masses of phosphates were beginning to come away. One month after the operation the prostate had diminished so much in size that a metal catheter with an ordinary curve could be passed without difficulty, and the shaft of it could be felt through the rectum for the whole of its length. The urine was acid, with only a faint cloud of mucus in it; every now and then soft masses of phosphatic material about the size of

a pea were passed down the urethra, causing severe pain; and when the sound was passed much more of the same material could be felt. The size of the bladder had increased a little; on several occasions it had retained four ounces of fluid, and the urgency and pain were much less severe; but micturition, as might be expected, was still very frequent day and night. So far as his general health was concerned, the patient had decidedly improved. He was looking much better in the face, and was much more comfortable. Instead of going as rapidly as he could downhill, he was holding his own well, and was able to look after his business. No morphia was required. He not unfrequently slept for two hours without being disturbed. The neck of the bladder was much less sensitive, and examination with a catheter did not cause anything like the degree of pain that it did before.

The second case was that of an enormously stout man, 64 years of age, who was sent to me by Dr. Houchin, of Stepney. The prostate was of very great size. There had been repeated attacks of cystitis and several of profuse hæmaturia. He was entirely unable to pass his urine, and had to introduce a catheter every hour night and day, each attempt being followed by intense pain. The bladder, which had been repeatedly washed out, was of fair size, and retained plenty of muscular power. Orchotomy was performed on November 20. There was no shock, but the abdomen was of such enormous bulk, and interfered with the respiration to such an extent, that the administration of the anæsthetic caused considerable anxiety. The patient rallied well, however, and for a week progressed favorably, sitting up in bed (he was quite unable to lie down, as even when sitting his abdomen seemed nearly on a level with his chest); the wounds were healing, and a small quantity of urine was passed naturally—the first time for fifteen months. On the eighth day, however, the patient had a fainting fit, the breathing became much embarrassed, and the urine, when it was drawn off by the catheter, contained a good deal of blood. Two days later the dyspnœa and the hæmaturia returned again, and though the patient rallied for a time under active stimulation, the respiration became more and more difficult, the face became agonised and covered with a cold perspiration, diarrhœa set in—the bowels acting eleven times in the last twenty-four hours,—and death ensued on the eleventh day after the operation. For two days previously a good deal of blood-stained urine had been passed at intervals by the urethra.

The immediate cause of death was failure of the heart. At the autopsy it was found exceedingly soft and flabby and loaded with fat. Masses of fat were found around the pericardium and attached to the margins of the lungs, which were markedly emphysematous. One kidney was cirrhotic to an advanced degree; the other contained many small cysts, but there was still a sufficient amount of secreting structure. There was no pyelitis. The bladder was large and thick-walled. In the interior was a calculus of considerable size, which had been lying in the pouch behind the prostate and so had escaped observation. On one occasion when the house surgeon, Mr. Langdon Down, had passed a soft catheter rather farther than usual into the bladder a sensation as if it rubbed against something rough had been experienced; but the calculus was quite inaccessible to an ordinary metal sound. There was also a certain amount of blood-clot, and the mucous membrane was intensely congested, roughened on the surface, and ulcerated in several places. The prostate, in spite of the venous congestion which had persisted for many days, was remarkably soft and flabby. The walls of the prostatic urethra were in contact with each other, but the forefinger could be passed down it from the bladder without the least difficulty. All parts of the gland were enlarged nearly uniformly. In addition there was an outgrowth projecting upwards from the vesical surface of one of the lateral lobes into the bladder; and this was soft, flabby, disc-shaped, instead of rounded, as such outgrowths usually are, depressed in the centre, and covered over with wrinkled mucous membrane—an appearance that could only be explained on the supposition that it had undergone a recent rapid diminution in size. A microscopic examination of several parts of the gland was made for me by Mr. Roxburgh, but there was no evidence of any difference from what is usually seen in sections of enlarged prostate.

I have brought these two cases before the Society because, though neither can be compared in point of success with my former one, an account of which was read before the British Medical Association in August last year, they both illustrate, as far as they go, the same fact that removal of the testes is followed invariably and rapidly by disappearance of the enlarged prostate In the first case the bladder had already been ruined beyond repair by cystitis and catheterism. Nothing could possibly restore the muscular fibres that had been destroyed, or give back to the walls the flexibility and

elasticity they had lost. But further progress was arrested: the kidneys, which were beginning to suffer from septic pyelitis, were saved, and the patient's condition was made infinitely more comfortable. In the other, death from heart failure—due no doubt, in part, to the operation, but probably, as no symptoms of it made their appearance for upwards of a week, even more to confinement to bed and general constitutional causes—ensued too soon for any definite microscopic changes to have shown themselves. But even in this one there was distinct evidence of a reduction in size of the gland and of a reopening of the prostatic urethra. In my first case, in which retention was the prominent feature, and in which it was impossible, owing to the shape of the prostate, to introduce a catheter, the route from the bladder became open, so that there was no residual urine, the patient being able to empty the bladder completely, and a catheter with an ordinary curve could be passed with ease.

There is, I imagine, no reason to fear that this operation will be in any way abused, as it is said oöphorectomy has been. The objections to it are too obvious, and patients from their age and sex are too well aware of what it means to them. But I believe, if the matter is laid plainly before them and the choice left to them, there is a very large number who would prefer the operation to a certain and lingering death by slow torture. And I am convinced, from the clinical results of the cases that have been operated upon, that we are perfectly justified in stating positively and definitely to them the result that will follow.

I must apologise to the Society for venturing to bring a new word before them. Castration is not a pretty term. Ὀρχότομειν is the Greek verb for performing the operation, and is classical. Orchotomy is the natural derivative from it. Orchectomy, which has been coined in America for the same proceeding, presumably on the analogy of colectomy and other similar words, cannot, I think, be justified on any other ground.

XXXII.—*A case of large Intra-thoracic Cystic Goitre causing Dyspnœa, and treated by operation.* By Anthony Bowlby. *Read April* 26, 1895.

S. G., æt. 34, a married woman, was admitted into Lucas Ward, St. Bartholomew's Hospital, on December 16, 1892, suffering from difficulty in breathing. She said that two years previously she first noticed a soft swelling in the episternal notch in the middle line of the neck, and that it had increased steadily and spread a little to each side. Ever since the swelling began she had suffered from shortness of breath, and during the last six months had had occasional attacks of transient but severe dyspnœa.

She had been born in London, and had always lived there. Her general health was good except for chronic bronchitis. She had been married eight years, but had never been pregnant.

The attack of dyspnœa from which she was suffering on admission was the most prolonged and severe that she had had. Her account was that she had got wet a few days previously, and had caught a bad cold; that at first she had pains in her limbs, cough, and the usual symptoms of nasal and pharyngeal catarrh, and that then the swelling in the neck had increased in size, and difficulty in breathing had commenced two days before she came to the hospital on the evening of the 16th.

The house surgeon, Mr. West, who admitted her, ordered hot fomentations to the throat, placed the patient in a tented bed, and kept a steam kettle in constant use; and these measures, together with the administration of some Squills and Tinct. Camp. Co., at once gave relief. The patient passed a restless night, but on the following day, the 17th, was more comfortable than she had been. At night-time she was again worse, and was quite unable to get any sleep; and next day, as she did not improve at all, I was sent for, and saw her at three in the afternoon, the dyspnœa having greatly increased since the morning.

I found the patient suffering from very severe dyspnœa. She was propped up in bed, and was quite unable to lie down; the breathing was stertorous and very laboured;

there was considerable recession of the lower ribs and epigastric region, and speech was difficult. She could swallow, but could only take very little nourishment at a time, and was much exhausted by the want of sleep and the struggle to get breath. Her temperature was 100·5°, but on the previous day it had been as high as 103°. Her respirations were about 50, and her pulse about 150 per minute. She had occasional attacks of coughing.

An examination of the neck showed at first very little swelling to account for so serious a condition. Such swelling as there was occupied the episternal notch, and caused a prominence in this region instead of a depression, and extended laterally under each sterno-mastoid. When the patient coughed, however, the swelling increased in a most extraordinary manner, and a large rounded mass was suddenly extruded from the chest into the neck, and then as suddenly disappeared. The way in which this tumour was projected reminded one much of the sudden protrusion of a large hernia during coughing. To the touch the swelling in the neck was smooth, rounded, and curiously soft; it appeared to be continuous with a rather enlarged left lobe of the thyroid gland, but the trachea in its upper part did not seem to be covered by any considerable thickness of tissue. The tumour seemed to move very slightly on deglutition.

An examination of the chest revealed a large area of dulness behind the sternum and cartilages of the first three ribs, and continuous with the normal cardiac dulness below. There were loud mucous râles in the trachea and bronchi.

It seemed clear that the patient was suffering from a large substernal goitre which was impacted in the upper opening of the thorax, and was compressing the trachea. It was considered that there had probably been a recent rapid filling of a cyst, and there was no evidence of the disease being malignant.

In spite of the urgent condition of the patient leave to operate at once could not be obtained, but an operation was permitted five hours later, and was performed at 9 P.M. with the kind assistance of Mr. James Berry, who saw the case with me at this time.

After a small amount of chloroform had been administered I made an incision from the thyroid cartilage to the sternum, and at once exposed the trachea above the thyroid isthmus, so that in case of need tracheotomy could be quickly performed. The skin incision was then extended obliquely

upwards to the left, and the sterno-hyoid, sterno-thyroid, and omo-hyoid muscles were divided, and a large cyst was exposed in the left lateral thyroid lobe. The cyst wall was exceedingly thin, and extended behind the sternum beyond view. Hoping that, if I freed the left lobe of the thyroid from above and laterally I might be able to draw upwards the intra-thoracic portion, I cleared it above and on its outer side, only to find that it was quite impossible to reach the lower margin of the cyst. At this stage, however, the breathing of the patient almost ceased, and I at once incised the cyst just above the sternum and opened the trachea below the cricoid cartilage. A feather inserted into the trachea caused the expulsion of some very viscid mucus, and after a very transient employment of artificial respiration the patient breathed quietly and easily through a large piece of rubber tubing passed down the trachea for several inches.

My attention was now turned to the cyst. It had already greatly collapsed, but it still contained several ounces of very clear and almost watery fluid. I opened it widely, and after evacuating its contents discovered that it passed into the thorax to a surprising extent. Its capacity was about a pint, or perhaps more. It had displaced the apices of the lungs laterally, and extended down to the base of the heart. Its walls were very thin, and were reflected over the large vessels, so that on looking into its cavity one saw the innominate carotid and subclavian arteries apparently uncovered by any structure. The arch of the aorta was similarly prominent, part of the cyst passing in front of it and part behind: the floor of the cyst rested on the base of the heart, the pulsations of which could be easily seen.

Now that the cyst was empty, it was seen that it was attached to the left lobe of the thyroid; but it was also evident that the gland, as a whole, was very slightly increased in size, the apparent enlargement being due to the pushing upwards and forwards of the left lobe by the distended cyst.

I did not consider that anything could be gained by removing either lobe of the thyroid, and it was evident that the cyst itself was irremovable. The operation was therefore completed by the washing out of the cyst with a solution of perchloride of mercury 1—4000, and by carefully stitching its walls to the skin around the episternal notch. The tube was then removed from the trachea, and the greater part of the tracheal incision was closed with sutures. Finally the cyst was dressed so as to shut it off as much as possible from

the tracheal wound, for it was obvious that suppuration might have caused great danger in a part so deeply situated.

The patient made an almost uninterrupted recovery. The tracheal wound closed in three days, and the cyst did not suppurate. The bronchitis gave a little trouble at first, and for a week after the operation the temperature ranged between 99° and 102°; afterwards it was normal.

The opening into the cyst closed in three weeks, and a month from the day of operation the patient left the hospital.

The case is of interest on account of the large size of the cyst, and the depth to which it extended within the thorax. In the latter respect the case is certainly of a very unusual character, for although "substernal goitre" is a recognised variety of bronchocele, it is well known that the part of the tumour behind the sternum is seldom large. The history also illustrates well the tendency that all cysts near to the air-passages have to increase in size when there is an acute catarrh of the trachea or bronchi. I have observed this tendency in hyoid bursæ as well as in thyroid cysts, and in the present case there seems no room for doubting that an increase in the contents of the cyst, combined with an attack of bronchitis, brought about the severe dyspnœa from which the patient suffered.

With regard to the treatment, I think it may be said that the opening and draining of the cyst were the only surgical measures that could be adopted. No doubt the removal of the cyst wall would have been more satisfactory had it been possible; but, considering its close attachment to the large vessels, it was evident that no such removal could be attempted,—the base of the cyst, indeed, was quite beyond the reach of the finger. There seems the less cause for regret at the present time because there is no sign of refilling the cyst, and the patient remains in good health.

XXXIII.—*Case of an infant in whom some of the Abdominal Muscles were Absent.* By R. W. Parker. *Read May* 10, 1895.

JAMES B., a newly born infant, was admitted into the East London Hospital for Children under the care of my colleague, Dr. Eustace Smith, whose permission I have to record the case.*

Though a full-term infant, it was very small and ill-nourished, but it took the mother's breast. The skin was rather jaundiced, wrinkled, and inelastic, the movements feeble, the cry almost inaudible, the weight 5½ lbs. The abdomen was large and very flaccid; as the child lay on its back the flanks bulged outwards markedly, owing to the weight of the unsupported viscera. The outlines of the intestinal coils could be clearly seen, and the outlines of the abdominal organs easily felt. When the infant cried, and also after a full meal, the abdomen became "blown out" to an unusual extent.

The abdominal wall was as thin as parchment and unresisting. Along the middle line, where the rectus muscles should be found, there was a little more resistance than over the lateral regions, and towards the lower attachments some rudiments of these muscles could be felt. The oblique and transversalis muscles were apparently quite undeveloped. On the other hand, the muscles of the back appeared to be normal.

The muscles of the extremities, though small, appeared to be normal.

The umbilicus was not depressed; it was normal as to position, but resembled a surface scar. In appearance the integument everywhere appeared normal.

Temperature.—

1st. M. 98°; E. 100°.	6th. M. 100°; E. 99·2°.
2nd. M. 100°; E. 98·4°.	7th. M. 97·8°; E. 98·4°.
3rd. M. 102°; E. 100·4°.	8th. M. 98·4°; E. 97·8°.
4th. M. 100·6°; E. 100·8°.	9th. M. 100·6°; E .104·6°.
5th. M. 99·6°; E. 99·8°.	10th. M. 100·8° (2 A.M.); 103·8° (10 A.M.); E. 103°.

* These notes refer to a case which I hoped to have exhibited to the members on the Clinical evening, November 9, 1894. The infant, however, was too ill to leave the hospital, and died next day.

Death resulted from collapse and emphysema of the lungs. An autopsy was made by Mr. A. T. Rake, the Surgical Registrar, whose notes I append.

Abdominal wall.—Behind.—Right side: latissimus dorsi slightly developed; external to it, arising from the eleventh and twelfth ribs, is a strand of muscular fibres passing obliquely downwards and forwards to be attached to the crest of the ilium. This band measures $\frac{3}{8}$ inch across in the middle, $\frac{5}{16}$ inch above, and about $\frac{1}{4}$ inch below. On turning back the latissimus dorsi there is seen a distinct posterior margin to this band of fibres, which represents the external oblique The erector spinæ on this side is well developed, and projecting beyond the erector spinæ the quadratus lumborum is seen, which appears in every way normal; it arises from the transverse processes of all the lumbar processes, and passes down to be attached to the crest of the ilium. It is about $\frac{1}{4}$ inch in breadth. The outer margin of the external oblique is semilunar in shape, and thins off so as not to be so well marked as the posterior border. A layer can be dissected off representing the fore-part of the rudimentary external oblique, exposing a deeper layer arising from the eleventh rib beyond the twelfth, and passing downwards and backwards to be attached to the crest of the ilium. On removing this layer, which represents the rudimentary internal oblique, the peritoneum is exposed, covered by the extra-peritoneal tissue. There is no trace of any muscle representing the transversus abdominis. The last dorsal nerve passes through the deep layer of muscle.

Left side: there is a slight amount of muscle representing the latissimus dorsi. On this side is a similar band of muscle to that seen on the right, measuring $\frac{1}{4}$ inch above, $\frac{3}{8}$ inch in the middle, and $\frac{1}{4}$ inch below. It is similarly split into two layers, the last dorsal nerve passing through the fibres of the deep layer.

In front.—There is a thin layer of muscular fibres passing from the cartilages of the ribs down to the level of the eighth costal cartilage, where there is apparently the first linea transversa. This body of muscle is well marked on the right, whilst on the left it is but faintly seen. Further down there is the merest trace of any muscular fibres representing the rectus on either side.

The chest cavity seemed very small as compared with the large abdomen.

Lungs.—Right: middle lobe is almost completely atelectatic.

Left: base of the upper lobe and the upper part of the lower lobe are completely atelectatic; all these undistended portions of the lung sink in water. The anterior edge of the left upper lobe and the inner surface of this lobe show much emphysema—some of the air-cells were extremely dilated, to the size of a haricot bean. The walls of these very distended alveoli were quite translucent.

Stomach and intestines.—Appeared quite normal.

Urinary organs.—The bladder was greatly hypertrophied, the wall measuring $\frac{3}{8}$ inch in thickness; it was situated wholly within the abdominal cavity. There was no obstruction to the urethra; the prepuce could be retracted over the glans. At the apex of the trigone was a depression, the bladder wall being here closely attached to the wall of the rectum. There was, however, no communication with the rectum. The ureter and pelvis of the kidney on each side were very greatly dilated and hypertrophied. The openings of the ureters into the bladder were quite free; there was no obstruction from calculi or other source in the ureter. The kidneys were large, and on section looked yellowish white.

Testes.—On each side the testis was situated in the iliac fossa, quite free from the internal abdominal ring.

XXXIV.—*Two cases of Gastric Ulcer treated by Laparotomy.* By L. A. Dunn. *Read May* 10, 1895.

SOPHIA LOUISA D., æt. 15, was admitted into Miriam Ward on May 19, 1894, under the care of Dr. Newton Pitt, suffering from abdominal pain and vomiting.

It seems that since last Christmas she had been a good deal troubled with gastric disturbance, especially after taking food. The pain was chiefly referred to the epigastrium. She had vomited to a slight extent occasionally about ten minutes after a meal, the ejecta being acid and free from blood.

On Wednesday, May 16, at 9 A.M., whilst at work, she was attacked with severe pain under the heart, and was carried up to Guy's Hospital. She returned home feeling better. The next day, May 17, the patient remained fairly well, but the pain persisted.

On May 18 she was seized with very severe pain all over the stomach, and vomiting. Castor oil was administered without effect; she passed a restless night, and was admitted into the hospital on the next day.

On admission the girl is well nourished, with a sallow, anxious face, dark eyes, and slightly anæmic lips. Temp. 101°, pulse 150, and respirations 33 per minute.

The abdomen is rather full, and moves little with respiration. It is not tense. Superficial tenderness is complained of over the greater part of the abdomen, but chiefly in the left hypochondriac region. There is slight fulness with increased resistance in the epigastric region and slightly to the left. The liver dulness on the right side is normal, but absent in the epigastrium.

The greater part of the abdomen is resonant on percussion, and the flanks are not dull; but there is a hyperresonant area occupying the left side of the epigastrium, extending halfway up the sternum, and reaching nearly to the left nipple laterally. On auscultation no rub can be detected, and the heart's sounds, which are transmitted through the tympanitic area, are apparently normal. A remarkable bruit of a bell-like tone can be heard synchronous with the heart's systole in the middle line of the abdomen, midway between the umbilicus and the ensiform cartilage.

The rectum is empty and ballooned. The tongue is furred. Urine, sp. gr. 1025, acid, pale, contains a slight trace of albumen. Menstruation has not yet taken place.

The diagnosis of local peritonitis in the region of the stomach, probably due to perforation of a gastric ulcer, was made by Dr. Pitt, and nutrient enemata ʒiv were ordered every three hours—after previously emptying the rectum by means of a simple enema,—and morphia gr. ¼ and atropine gr. $\frac{1}{100}$ every four hours subcutaneously.

Later in the evening the report states that the patient has not been sick, and the pain has been subdued by the morphia. The simple enema was very efficient, and the nutrient enemata are retained. The abdomen is still rigid. Temp. 102·4°, pulse 144, resp. 40 per minute.

May 20.—Patient has the "abdominal expression," and is anxious for a little milk to drink; the mouth is very dry. The abdomen is not distended, but is slightly more rigid. The pain has not diminished. She was unable to void urine till hot cloths were applied. The tympanitic area, supposed to be due to some collection of gas, has diminished somewhat in size, reaching as far as the right edge of the sternum. The tympanitic note is not so resonant as it was yesterday. The whole abdomen is resonant. The stomach resonance reaches apparently to one inch below the umbilicus. Patient complains of deep-seated pain in the left hypochondriac region about the sixth and seventh cartilages. There is cutaneous pain both in the left hypochondriac and iliac regions.

The recti are rigid, more so than yesterday, and the whole abdomen is rounded and tense. The upper part of the abdomen moves more, possibly owing to deeper respirations.

Temp. 100°, pulse 128, resp. 24 per minute. The pulse increased in rapidity during the day, and showed marked dicrotism.

The morphia almost entirely relieves the pain. There is a tendency to draw up the knees.

11 P.M.—The patient is decidedly worse to-night, rather in the matter of degree than in the appearance of fresh symptoms, but is not at all collapsed.

Mr. Dunn was called in at 2 A.M., and decided to explore the abdomen at once. A port-wine enema was given a quarter of an hour before the operation.

Having taken precautions to avoid sepsis and shock, the

A. C. E. mixture was administered, and a median incision made just below the umbilicus. The peritoneum was rapidly reached, and opened after all bleeding had been arrested. On exploring the abdominal cavity with the finger, the stomach was felt to be fixed on to the liver, which was itself also attached by adhesions to the anterior abdominal wall. Three or four chromic gut sutures were passed through the muscular and peritoneal layers of the margins of the wound, and their extremities held together by means of clip forceps. By pulling on these the margins could be approximated, whereas the wound might be widely opened by slackening them. The wound was now enlarged upwards by means of scissors towards the ensiform cartilage, thus making the incision about five and a half inches long.

On separating the adhesions which fixed the liver to the abdominal wall a quantity of opalescent fluid escaped. The liver was then pulled upwards and the anterior wall of the stomach pushed backwards, and now it was that some brownish fluid, like weak coffee, containing gas bubbles and one or two small masses of coagulated milk, escaped. It welled up from a depth at the left of the incision, and was found, on subsequent examination, to be strongly acid and to contain a little albumen. Several more adhesions were broken down, but still no perforation could be seen, and it was only when the sutures were slackened and the left margin of the wound pulled outward to the utmost, whilst steady traction was made upon the stomach towards the right, that the hole in this viscus became visible.

This was an opening, oval in shape, about one third by a quarter of an inch in size, with white granular edges, and a periphery of inflamed stomach wall with flaky lymph covering it. The opening was then brought up as completely as possible into the wound, and closed by means of two rows of fine silk Lembert's sutures. The peritoneal cavity was flushed out thoroughly with warm boiled water, and an attempt was made to turn up the great omentum over the anterior surface of the stomach, in the hope of thus strengthening the weak spot. This could not be accomplished owing to the crumpled and contracted condition of the omentum, the result of a somewhat lengthy exposure.

The abdominal wound was closed with a deep row of chromic gut sutures, including muscles and peritoneum, and a superficial row of silkworm-gut sutures, taking in skin only. Iodoform was dusted over the wound, which was dressed with

sal-alembroth gauze and wool, the whole being retained in position by means of a many-tailed bandage.

The operation lasted one hour. After the operation a port wine enema was given, and a subcutaneous injection of morphia and atropine half an hour later. This was repeated at intervals of four hours. Nutrient enemata were ordered every four hours, together with one injection of half a pint of warm water.

May 21.—Patient is comfortable. Tongue furred and dry. She has passed 22 oz. of urine.

May 22.—There have been no bad symptoms. The atropine was omitted from the injections, as she became slightly excited. Temp. 99°, pulse 116, resp. 10 per minute. There is tenderness on pressure over the area of the stomach. Mackenzie's continuous feeder was tried, but did not prove very satisfactory. One ounce of warm water was given by the mouth every two hours, which was greatly enjoyed and produced no sickness.

May 23.—Is quite comfortable. The injection of morphia was diminished to $\frac{1}{4}$ gr.

May 24.—Feeding by the mouth was commenced. It consisted of 1 oz. of peptonised milk, with a drachm of a two per cent. solution of hydrochloric acid, every two hours. After the second dose she complained of nausea and flatulence.

May 25.—The temperature fell to-day to 97·2°, but rose in the evening to 99·8°. She was still nauseated with the peptonised milk, so milk and soda water was given, and was promptly returned. Warm water alone was tried for the next three hours, after which 2 dr. of peptonised milk was added. She retained this.

May 26.—Patient was sick this morning, bringing up half a pint of greenish fluid with mucus. She has slept badly, and hiccoughed at times. Pulse 112 per minute. The wound was dressed. The dressings were soaked with a serous discharge, and the wound had gaped very slightly at the umbilicus. There is no distension of the abdomen.

May 27.—Peptonised milk has been omitted. There has been no nausea since. The water given by the mouth now amounts to 2 oz. every two hours. The enemata as before.

May 28.—The wound was again dressed; it had gaped slightly in places. Re-dressed with cyanide gauze.

May 30.—Soda water and milk, of each an ounce, have been taken every two hours. It caused neither pain nor nausea. Temp. 99·8°, pulse 60, resp. 20.

May 31.—The rectum was very foul, so it was washed out with boracic lotion, and an iodoform suppository inserted. The milk was increased to 2 oz. every two hours, and beef tea added. Nutrient enemata of 4 oz. given every eight hours.

June 2.—Last evening the patient complained of slight pain, which, however, passed off. At ten this morning she seemed comfortable; pulse 88 per minute. At 1 P.M. she vomited twice, and complained of great pain in the abdomen; the pulse ran up to 136 per minute. Stomach feeding was stopped. At 2 P.M. the pain was much worse; pulse 160. A morphia and atropine injection was given. At 4 P.M. Mr. Dunn saw the patient. She was complaining of great pain, the face was drawn, and the eyes sunken. She was very restless, with a rapid feeble pulse. The wound was dressed and found to be healthy. Over the region of the stomach there was a tympanitic note, which extended up to the fifth rib. Her condition was so grave that it was decided to have another consultation at 5.30 P.M. 5.30 P.M.—She is more comfortable; pulse 120. The pain is not so intense. The tympanitic area has not increased. The heart's sounds are well transmitted across this area. Bruit d'Airain can be heard. The morphia is to be omitted. Nutrient enemata are to be given every six hours, and 1 oz. of water by the mouth every two hours. At 9 P.M. another consultation was held. The tympanitic area had increased upwards, and reached to the fourth rib. The pain was most severe and paroxysmal in character; it was felt all over the abdomen, which was very tender, but moved fairly well. Pulse 120. Her condition was such that both Dr. Pitt and Mr. Dunn were of the opinion that she would not live through the night if nothing were done; hence at 10 P.M., having taken the usual precautions against sepsis and shock, an incision 3 inches in length was made along the left costal margin, and the abdominal cavity was rapidly opened. On exploring with the fingers it was found that the stomach was fully distended, and adherent to the parietal peritoneum on the left of its anterior surface by a few recent adhesions. These easily separated, but no gas or fluid escaped. The finger was next passed to the right, towards the site of the former operation. The adhesions here were found to be perfectly secure. The abdomen was closed in the same manner as before, and the wound was dressed with cyanide gauze. The whole operation lasted only twenty minutes. The patient recovered from the anæsthetic, and had

a morphia injection at 11 P.M. This was repeated at 2 A.M., together with three minims of Liq. Strychninæ.

June 3.—Seems much better, and has not been sick. Has required no more morphia.

June 9.—Has been going on perfectly well. The wound was dressed, and the superficial stitches were removed. It had healed completely. She takes the whites of three eggs, a drachm of brandy, and a pinch of salt in a pint of warm water, one ounce every two hours by the mouth.

June 14.—Is now fed entirely by the mouth, and experiences no discomfort.

June 20.—Patient sat up to-day. She is still pallid, but very cheerful.

July 2.—Left the hospital for a convalescent home.

The second case was that of Clothilda C., æt. 28, who was admitted into Mary Ward on June 4, 1894, under the care of Dr. Hale White for pain in the abdomen and vomiting. She had previously been a patient in St. George's Hospital for a period of seven weeks, and had left much relieved. Her present trouble commenced three weeks before admission with vomiting half an hour after food, flatulence, palpitation, and severe pain in the epigastrium.

On admission the abdomen was superficially tender, most markedly so in the epigastrium. There was no distension. The bowels were costive, and the temperature 99·2°.

Under medical treatment she progressed favorably till June 22, when after eating some custard she got up in the afternoon for about one hour. The pain in the abdomen now returned with great severity, and she was obliged to get back to bed. She vomited after taking any nourishment, even small doses of peptonised milk; hence on June 24 it was thought wise to feed her entirely by the rectum, a nutrient suppository being alternated with a nutrient enema every four hours. Subcutaneous injections of morphia were given occasionally to relieve the pain. On June 29 the patient was very restless, complained of a good deal of pain, and vomited slightly. At 6 P.M. she screamed out with most violent pain in the epigastrium, and vomited half a porringer-full of greenish fluid. She became very collapsed, and the pulse was rapid and thready. As she did not improve, the house physician sent for Dr. Hale White. When he saw her he at once asked Mr. Dunn to meet him. It was now midnight, and Dr. Hale White and Mr. Dunn found her then

bathed in perspiration, and groaning with pain. Her face was pinched and her eyes sunken. Her extremities were cold, clammy, and of a dusky hue. The pulse was very rapid, and scarcely perceptible at the wrist. She was very restless, tossing her arms about, and throwing off the bedclothes. The abdomen was rigid and tender.

As there seemed to be no doubt about the diagnosis of perforated gastric ulcer the patient was wrapped up in hot cotton wool and blankets, and placed on the operating table. Ether was administered, and with all aseptic precautions a median incision was made extending from the ensiform cartilage towards the umbilicus. The peritoneum was quickly opened and the stomach exposed. This viscus was distended with a fair quantity of gas, nothing abnormal could be found on its surface, and although the fingers explored the whole of the anterior aspect no adhesions were encountered, and not a trace of gas or fluid escaped from the wound. Neither did it seem likely that there was any collection of fluid behind the stomach, since the pulsations of the aorta could be so readily felt transmitted through the walls of the stomach. As the only abnormality revealed by the operation was a trifling adhesion of the great omentum to the parietal peritoneum, and as the patient was very collapsed, it was decided to pursue the investigation no further, and to close the wound. This was carried out on the same plan as in the former case.

She recovered from the shock of the operation, but continued to vomit the same bile-stained fluid. The restlessness and pain, too, continued much as before the operation. She was kept under the influence of morphia with rectal feeding as before, together with an occasional large enema of warm water. She became weaker, and died in her sleep on the fourth day after the operation. The wound was healing and in no wise gaped, despite the persistent vomiting. Dr. Newton Pitt made the post-mortem examination fourteen hours after death, and reports that the abdominal incision had united well along the outer stitches; the inner peritoneal surfaces, however, were not so uniformly and firmly adherent, but nowhere was there anything like a trace of suppuration. The peritoneal surface of the intestines was healthy, and on lifting up the omentum, which adhered to the abdominal wall and was slightly thickened, nothing at first was seen to be abnormal. The stomach was found to be adherent to the under surface of the liver by a long loose fold of peritoneum

an inch and a half long, situated to the left of the falciform ligament.

When examined, the peritoneum and subjacent tissue was seen to have undergone gastric digestion for an area 2 inches across, and through the softened tissue fluid exuded from a large ulcer on the anterior wall of the stomach. When the stomach was opened this was found to lie with its long axis across the length of the stomach, and near the smaller curvature; length $\frac{7}{8}$ and breadth $\frac{1}{2}$ inch.

There was no induration of the edge, and the base appeared to be formed solely by the peritoneal surface, which had by this time become diffluent. There was rather more than an ounce of fluid in the stomach, and this was bile-stained. No fluid at all was found in the peritoneal cavity, and no acute inflammation. No extravasation of recent lymph. The long peritoneal adhesion to the liver was tough and not very recent.

There was nothing of importance discovered in the rest of the viscera.

In reviewing these two cases the first thing that strikes one is that symptoms very like those produced by perforation of a gastric ulcer may be present without this having actually occurred.

In the first case, on the thirteenth day after the operation severe symptoms suddenly appeared, which were almost identical with those exhibited by the patient on her admission into the hospital; so much so that both Dr. Pitt and I were strongly of the opinion that a fresh perforation had taken place. We, however, delayed operating as long as possible, being very averse to opening up the abdomen a second time, and it was only at our last consultation, when the patient's condition was so grave as to make us fearful of her not surviving the night unless something were done, that we decided to operate, and found nothing save a distended stomach dragging upon some recent adhesions.

In the second case it is doubtful whether perforation had happened or no.

Certain it is that nothing of the kind was discovered at the operation,—and even if it had existed then, there had not been the slightest escape of the gastric contents—as verified not only at the time of operation, but also at the subsequent post-mortem examination; still, I think, in the mind of any who saw this patient on the night of the opera-

tion, not a shadow of doubt existed that perforation of a gastric ulcer had taken place.

The operation that was performed in the first case was followed by immediate relief; whilst that in the second case, if it did not retard, I believe it did not accelerate the fatal termination.

What the actual cause of death was is difficult to determine, but, considering that the patient had subsisted upon nutrient enemata for twelve days, and had been almost constantly vomiting for the last week of her life, exhaustion may lay claim to an important share in the result.

The reasons the perforation was simply stitched up without excision of the ulcer were that it occupied much less time, that the wide-spread induration of the anterior wall of the stomach showed that the ulcer was large, and for its complete removal would have required extensive mutilation of the stomach; and that, owing to the difficulty of getting the aperture into the wound, it would have been almost impossible to have securely sutured so wide a gap.

In conclusion I must acknowledge my indebtedness to my colleagues, Drs. Hale White and Newton Pitt, for permission to publish these cases, as well as to Mr. C. A. Peters, not only for his very accurate report of the successful case, but also for the unwearying attention he bestowed upon the patient in the capacity of dresser, to which in great part must be attributed the favorable issue.

XXXV.—*Two cases of Perforated Gastric Ulcer: in one case excision of edges of ulcer and suture, death; in the other formation of reparative adhesions, laparotomy and drainage, recovery.* By A. Quarry Silcock. *Read May* 10, 1895.

M. H., æt. 24, a barmaid, was admitted to St. Mary's Hospital under the care of Dr. Lees on April 10, 1894.

Up to a fortnight before admission she had enjoyed good health. Whilst standing at the bar on March 24 she was taken with pain in the stomach coming on suddenly. It was not so severe as to interfere with her work, but was slightly aggravated by taking food: she did not seem to have thought much of it. On the 7th of April, whilst standing at the bar after her supper, she was attacked by severe shooting pain in the pit of the stomach—so severe that it made her scream. She left her work and went to bed immediately. She did not vomit after the onset of the pain, nor did she feel sick. The pain was increased by drawing a deep breath, or coughing, or by any attempt to sit up. Next day her temperature was 103°, she said.

The following was her condition on admission:—She lay in bed on her back with her legs drawn up. She could turn on to her right side, but not on to her left. She spoke in a low voice, and was obviously very ill, but did not appear to be in great pain. The respiration was increased in frequency; the pulse 124 per minute. The abdomen was not tumid, but the left rectus muscle was tense, and there was great tenderness on palpation, especially in the left hypochondriac and epigastric regions. The diaphragm did not move in respiration. To light percussion the liver dulness was obscured by a tympanitic note as far outwards as the right nipple line. On the left side in front there was tympanitic resonance as high as the nipple, which extended outwards to the axillary line, rising as high as the ninth rib; over the ninth, tenth, and eleventh ribs in this situation there was great tenderness. Over the left back the resonance was normal to the level of the angle of the scapula, below which there was a zone of dulness. On deep percussion this dulness gave place to a

"boxy" note; three fingers' breadth below the angle of the scapula the resonance was tympanitic. On placing a coin over the margin of the thorax in the left nipple line and tapping it with another the bell sound was well heard to the right of the middle line, and on the left side as high as the nipple, but not in the middle line; it could be made out over the lower part of the left back, but not so well as anteriorly.

After consultation with Sir William Broadbent, Dr. Lees asked me to open the abdomen.

An incision was made three inches long immediately below the left costal margin, and chiefly through the left rectus muscle. On opening the peritoneum some bubbles of gas and clear fluid neutral in reaction escaped. No particles of food were mingled with this fluid. The stomach was partially drawn out through the wound, and its exposed surfaces carefully scanned. Eventually, amidst the remains of recent adhesions of lymph which feebly glued the viscus to the anterior abdominal wall and had been broken down in the necessary manipulations, there appeared a small perforation admitting the end of a large probe: it was in the anterior wall, midway between the upper and lower borders, and the cardiac orifice and the pylorus. The edges of this small perforation were yellow and necrotic: they were cut away and the freshened surfaces of the wound brought together by a silk suture passed through all the coats of the stomach: thus the tendency to eversion of the mucous membrane was overcome. Then the peritoneum around the wound was infolded by means of three Lembert's silk sutures. A small drainage-tube was so placed as to carry away any discharge from the neighbourhood of the perforation, and stitched to the margin of the parietal wound—the latter being closed in the usual manner, and dressed with iodoform, cyanide gauze, and wool.

It was not thought necessary to wash out the abdomen because what little discharge had collected escaped when the peritoneum was opened, and there were no signs of a general peritonitis. The stomach and parts involved in the operation were sponged over, and dried by means of sponges which had been soaked and wrung in 1 in 1000 sublimate solution. The subperitoneal lymphatic spaces had probably become infected, for continuous lines and patches of exudation involved the peritoneum and the subperitoneal tissues covering the anterior edge of the left lobe of the liver, and the upper and under surfaces of the organ in its immediate neighbourhood. It was clearly impossible to get rid of

infection here unless the parts had been freely scraped or by the application of strong escharotic antiseptics. Particular attention is drawn to this condition because herein possibly lay the explanation of the fatal issue of the case.

The patient was fed by nutrient enemata, nothing being given by the mouth until the second day, when ʒj of water was allowed every hour. On the 14th ʒj of rice (which had been boiled for six hours, then rubbed through a hair sieve) mixed with strong beef-tea was ordered to be given every hour by the mouth. Up to the 19th the patient improved in health, but during the whole of this time there was fever, and not until the seventh day after the operation was the temperature normal for a few hours continuously. The pulse, too, although its frequency lessened after the operation, did not fall below 100 per minute, and often was 110 per minute, or more. Thus we were clear that the patient was septicæmic, although the discharge from the wound was slight, and there had been no suppuration. She presented no sign or symptom of general peritonitis; her diaphragm now moved normally in respiration; the abdomen was nowhere tender. A marked feature in this patient's case was constant thirst and great restlessness; with the object of lessening the severity of the former symptom rather more liquid food was given at this time by the stomach, but on April 20 the temperature rose to 101·8°, and it was deemed advisable again to stop all food by the mouth. When the dressing was changed in the evening a thin whitish turbid fluid welled up into the wound; it had a fœtid odour, and was very faintly acid in reaction. This sero-purulent discharge continued, so that the dressings had to be changed pretty often. The patient, too, became weaker, the fever increasing. Every effort was made to drain the subphrenic region, but without avail. On the 24th of April she was much worse, clearly in a dying condition; "no sickness or abdominal pain," runs the clinical clerk's note, "but pulse weaker and intermittent. Blood passed by rectum, red in colour, probably due to irritation by enemata." Pleural friction was heard at left base. That afternoon an anæsthetic was administered, and an endeavour was made to drain the subphrenic region by means of an opening in a line with but posterior to the parietal wound; in doing so the left pleura was opened at its extreme lower periphery. The thoracic wound was immediately closed and the attempt abandoned. On the 25th of April the patient died, clearly from septic absorption.

Report of autopsy on the body of Mary Ann H—(April 26, 1894).—Body of a very anæmic young woman of about twenty years. Two surgical incisions, one just below the left costal margin, another behind the anterior axillary line in the ninth intercostal space.

On opening the thorax the diaphragm was seen to be displaced upwards, reaching on both sides to the fourth space in the nipple line. The parietal pleura was punctured opposite the second of the two surgical incisions mentioned above. The puncture was apparently healing, though the diaphragmatic surface of the left lung was covered with puro-lymph, which formed an adhesive layer between lung and diaphragm.

The lungs and heart presented no noteworthy feature save the lymph on the under surface of the left lung.

The middle part of the abdominal wound was still open, and through the opening pus could be seen. On further examination the stomach was found to be immediately beneath the wound, and this part of the organ was found to present a raised plate of firm lymph in which were embedded several stitches. The stomach was attached to the abdominal wall by old fibrous adhesions which extended all round the region in which the incision had been made in the abdominal wall; the incision lay entirely within this zone of adhesions.

The edge of the left lobe of the liver was firmly adherent to the stomach.

Subphrenic abscess.—During the manipulation of the stomach a great deal of pus welled up from a large subphrenic abscess, which was bounded above by the diaphragm, below by the upper surface of the left lobe of the liver and the anterior surface of the stomach. On the right the abscess was bounded by the falciform ligament, and on the left it extended to and surrounded the spleen. Below the abscess cavity was bounded by the spleen-shelf (costo-colic fold), to the left and to the right by the adhesions between the stomach and the abdominal wall. The rest of the peritoneal cavity, including the lesser sac, was absolutely normal in appearance.

Stomach.—On opening this viscus an ulcerated spot ¾ inch by ½ inch was found to be closed by the sutures mentioned above; in the depths of the ulcer was found a deep stitch, which lay loosely. A section through the ulcer showed it to be quite firmly closed.

In the wall of the stomach were two scars from old healed ulcers—one in the fundus, another in the pylorus.

The large ulcer, described above as having been sutured, was situated 3¾ inches from the cardiac orifice and 1½ inches below the lesser curvature. The kidneys were normal save for cloudy swelling.

I do not know what more I could have done. Here was a fairly healthy young woman, a recent perforation, and that small in size, and easily get-at-able; the surgeon requisitioned at the earliest possible moment; the operation of closing the perforation perfectly successful. Yet the perforation had existed a sufficiently long time before operative interference to allow infection to reach the subperitoneal lymphatics and to become generalised; it could not have been evicted, even locally, by any amount of washing; hence the condition of things found at the autopsy.

Let this case of fatal issue be contrasted with the following:

A. P., æt. 24, cook, was admitted into St. Mary's on April 7, 1894, under the care of Dr. Cheadle.

Five days previously she had been feeling unwell all day, and after going upstairs in the evening she was sick and fainted; on recovery she found that she had brought up a large quantity of blood. After admission she complained of occasional severe pain over the whole abdomen and much epigastric tenderness. The diaphragm did not move in respiration, the epigastric abdominal wall receding during inspiration. She complained of pain on deep inspiration. She was treated by Dr. Cheadle for acute ulceration of the stomach. On April 14 (seven days after admission) she was seized with sudden acute pain in the abdomen, which was not localised to any one spot. She vomited and ejected her last meal, but no blood. The pulse was 120 per minute; she was blanched, and cried out for relief from the pain. Soon she vomited again, her condition becoming one of collapse. On examination of the abdomen it was noticed that the diaphragm, which had recovered its function to some extent, was now motionless, whilst the right half of the epigastrium was somewhat bulged. On percussion the area of liver dulness was replaced by a tympanitic note. The upper limit of the tympanitic area began on the right side at the lower edge of the fourth rib in the nipple line, passing outwards horizontally into the axilla; and to the left of the middle line followed the lower

limit of the cardiac dulness; this was raised an interspace, the apex-beat being felt in the fourth space a little inside the nipple. A bell sound could be obtained over the tympanitic area, most markedly a little to the right of the ensiform cartilage, but decreasing in loudness centrifugally from this point. The tympanitic note was lost a few fingers' breadth above the umbilicus, the lower portion of the abdomen being less tympanitic. I saw the patient with Dr. Cheadle, and we agreed to wait a few hours before interfering surgically since the patient was so much collapsed. In the evening I opened the abdomen by an incision four inches long made close to the margin of the thorax, its lower end being an inch to the left of the middle line. A small quantity of turbid serum of neutral reaction escaped on opening the peritoneum, together with a few bubbles of gas. A wide band of fibrinous adhesions bound the anterior surface of the stomach to the under surface of the left lobe of the liver; bordering the limit of the adhesions was a zone of injected vessels lying in the anterior wall of the stomach. The under surface of the left lobe of the liver, its anterior edge, and the under surface of the left half of the diaphragm were flecked with lymph and patches of exudation, which lay partly on the peritoneal surface, partly beneath it. The stomach was semi-dilated: the exposed portion seemed to be quite normal elsewhere than at the site of the perforation. A drainage-tube was inserted and the wound closed by silkworm-gut sutures.

This patient made a slow but a practically uninterrupted recovery: the wound had completely healed on April 26, and she went back to the medical wards on April 30. She has suffered since from impaired locomotion of the stomach, and has been from time to time under treatment since that date as an out- or in-patient.

It is as difficult to account for the successful issue of this case as it is easy to explain the fatal result in the first. The latter was in a far better condition than the second patient, who indeed at the time of the operation seemed to be *in extremis*. At the outset, however, the fatal case had much fever, whilst the second had but slight septic fever before the laparotomy and less subsequently; in fact, I have been tempted to think that recovery might have ensued without surgical aid.

One lesson I have learnt from the successful case is the inadvisability of interfering with adhesions if these appear

to shut off the perforation from the general peritoneal cavity. Had I interfered with them I should have exposed a perforation, no doubt, but possibly a perforating ulcer of a magnitude which would have been difficult or impossible to deal with adequately by excision and suture; and without doubt I should have opened up the peritoneum to widespread infection.

I have to thank Dr. Cheadle and Dr. Lees for permission to publish these cases, and for their kindly assistance and advice in the treatment; Dr. J. L. Morton for the report of Case 1; and Mr. J. J. Clarke, F.R.C.S., for the report of the autopsy of the same.

XXXVI.—*Extensive Degenerating Nævus of the Bladder.* By W. Arbuthnot Lane, M.S. *Read May* 10, 1895.

W. D., æt. 3½ years, was admitted into St. John's Hospital, Lewisham, under the care of Mr. Lennard Stokes in September, 1894.

When the child was three months old the mother noticed for the first time some nævoid growth about the anus and some minute patches scattered over the buttock. They gradually lost their red colour, and became bluish and firm. That about the anus is at the present time hard and nodular, as if it had undergone cystic degeneration. It does not extend into the rectum.

Two years ago the child commenced to pass bloody urine at times, while occasionally pure blood or blood-clot was discharged from the urethra. This discharge of blood accompanied straining in defæcation, and the mother was for a long time under the impression that the blood-clot escaped from the rectum. The child suffered much from constipation. For intervals of time the discharge of blood would cease, the longest period being from April to August, 1894.

Since her admission into the hospital on October 10 the urine had always contained blood, and during difficult defecation large flat clots and a quantity of fluid blood were passed. It seemed very probable that much of the violent straining was due to a forcible extrusion of the clot from the bladder, and not to any desire to defæcate. Mr. Stokes, in consultation with Mr. Burroughs, diagnosed the condition as nævus of the bladder, and as the child was very blanched and exhausted from the very considerable loss of blood, he asked me to see her with a view of operating. I saw her on October 22, when I exposed by the ordinary suprapubic incision an unusually large and firm bladder. On incising its anterior wall great masses of nævoid tissue prolapsed through the opening made. Most of this was soft and compressible, and bled from its surface when manipulated. On introducing the finger and separating the edges of the incision, the whole of the mucous membrane of the bladder, with the exception of two narrow strips where it was normal, was seen to be converted into large masses of the same appearance as that already described.

Some portions of the growth seemed more vascular and bled more readily than the remainder, which contained firmer lumps and was apparently undergoing a cystic degeneration. Feeling that I should probably do more harm than good by attempting to remove as much as I could by ligature, I determined to temporize and watch the course of events. Consequently the opening in the bladder was closed by sutures together with the skin incision. Hazeline was injected into the bladder for some days after the operation, and for a time the bleeding ceased.

Recently it has returned at long intervals, but in very small quantities. The general condition of the child is also very much better than it was before the operation. It is fat and rosy, and apparently in robust health. Should the hæmorrhage recur and cause anxiety, we propose to open the anterior surface of the bladder, having previously exposed its peritoneal aspect to avoid damage to that portion of its surface, and to remove as much of the growth as we can with the electric snare. At the same time we hope that by the progress of the degenerative change which appears to be now present we may be able to avoid further operative interference, bleeding, should it recur, being stopped by local applications. Mr. Stokes and Mr. Burroughs deserve all the credit of the diagnosis. I expected to find an adenomatous or papillomatous growth. As I have never heard of such a condition of the bladder, I thought it of sufficient interest to bring before this Society.

XXXVII.—*A case of Pylephlebitis with Abscesses in the Spleen: foreign body in mesenteric vein: death from pyæmia.* By Sidney Phillips, M.D. *Read May* 24, 1895.

W. G., æt. 52, warehouseman, was admitted into St. Mary's Hospital on September 11, 1894, with the following history:—His father died of phthisis; the patient himself had rheumatic fever at the age of seventeen; his general health had always been good, but he was subject to winter cough and asthma; there was no history of syphilis; he had at one time taken spirits freely.

Present illness.—On August 13 he was so far as he knows in his usual health, and went with his family to stay at Ramsgate. On the next day he went out in a brake, and, the evening turning out inclement, he thinks he got a chill. On the day after this, at about 5 P.M., while out for a walk, he was seized with a severe rigor lasting sixty-five minutes, after which he sweated profusely for two hours. After the sweating ceased he felt perfectly well, and went out again for a walk; but the rigors recurred on the succeeding four days, attacking him with great regularity on each day at 5 P.M.

He was treated by a medical man with quinine, and returned to London at the end of a week. After that the attacks of shivering diminished both in frequency and in severity, and for five or six days—that is, from the 1st to the 7th September—he was free from any attack. On the latter date he had another rigor lasting about half an hour, and on the advice of his medical attendant he came to St. Mary's Hospital.

On admission, on September 11, he was extremely pallid and anæmic; the temperature was 102°, the pulse 100 per minute. Nothing abnormal was found about the heart except that the impulse could with difficulty be felt; his respirations were 40 a minute, and he "wheezed" considerably on breathing; there was a cough and muco-purulent expectoration, with evidences of considerable emphysema and bronchitis. The left lung, below the angle of the scapula, gave a defective note on percussion, and over the same area the breath-sounds were rather weaker than on the right side, though accompanied by loud mucous râles.

His appetite was bad. The liver was much enlarged, its edge being felt in the vertical nipple line as low down as the level of the umbilicus; it was not painful or tender. The superficial veins of the abdominal wall were rather over-full. The spleen was very much enlarged, its edge reaching four fingers' breadth below the costal margin, and coming forwards as far as the tip of the eighth rib; it felt hard, and moved freely with respiration beneath the abdominal walls.

The urine was acid, of sp. gr. 1012, without any albumen or sugar.

When I saw him on September 12, the day after the above notes were taken by Mr. F. P. Piper, M.B.Lond., the house physician, and Mr. J. Hurst Smyth, the clinical clerk, I introduced an exploring needle into the left pleural cavity, and the syringe filled at once with ½ oz. of pure blood. During the next two days he continued to get worse, being delirious at night, with a pyrexia of 99° to 101°, and the mucus he expectorated was slightly reddened by blood. On the 14th, his respirations being 42 and his breathing becoming more distressing, I aspirated the left pleura and drew off 9 oz. of bright red blood. This, like the fluid previously withdrawn, coagulated immediately, and under the microscope showed nothing to distinguish it from pure blood.

The dyspnœa was relieved by this aspiration, and the liver became lessened in size; the enlargement of the spleen, too, *appeared* to be much lessened, so that finally it could not be felt through the abdominal wall. But he gradually lost ground, the fever, the nocturnal delirium, and the sleeplessness continuing. His gums became spongy, his breath smelt septicæmic, and he died on the night of September 17, just a week after admission into the hospital and thirty-four days from the onset of his illness. I may add that blood from the finger tip was examined twice during his illness, but there was only found a great decrease in the number of red corpuscles.

His treatment while in the hospital consisted of the administration of quinine, of expectorants and stimulants, and of aspirating the pleural cavity to relieve the dyspnœa.

At the *autopsy* made by Mr. J. J. Clarke, M.B., F.R.C.S., pathologist to the hospital, the *spleen* weighed 2 lbs. 6 oz., and in its substance were three large abscesses with sloughy limiting membranes; these abscesses destroyed almost the whole of the spleen, and what little splenic pulp remained was softened, congested, and rotten.

The *liver* weighed 7½ lbs. On cutting across the small omentum, pus poured out of the divided portal vein, and on section of the liver pus was found throughout its substance; but everywhere it was in the hepatic ramifications of the portal vein, and nowhere formed any localised abscess in the liver itself. On slitting up the portal vein and its branches in the downward direction there was found pus in the splenic vein, and in this vein close to its termination in the portal vein was a soft semi-puriform clot.

In the mesenteric veins there was also abundance of pus, and in one of the main branches of the inferior mesenteric vein were found two black bristles, which were probably originally parts of a single bristle. One part was about ¼ inch and the other ½ inch long, and they were similar to the bristles used in hair brushes. The ends of the bristles appeared just indented in the lining membrane of the vein, but there was no sign of any injury to the vein wall, or any trace of the passage of the bristle through the vein or intestine. The lungs showed a state of advanced emphysema, with much mucus in the air-tubes. The lower part of the left lung was much congested, and the left pleura contained about 1 oz. of blood.

The right pleural sac was quite obliterated by adhesions. There was nothing abnormal in heart or kidneys.

Remarks.—When this patient first came under my observation there was evident a very great enlargement of the liver and of the spleen, with bronchitis, left pleural effusion, pyrexia, and a history of rigors and sweats for four weeks, at first diurnal and later on at irregular intervals: there seemed little doubt that there was pus-formation somewhere, and when the fluid in the pleura was shown by the exploratory needle to be blood and not pus, it seemed almost certain that some part of the abdominal cavity was the seat of the suppuration.

There was no softening or fluctuation, however, detectable in either the enlarged liver or spleen, and I came to the conclusion that suppuration in the portal vessels existed from some unascertainable cause.

The autopsy showed a condition of suppurative pylephlebitis with abscesses in the spleen. It is improbable that the splenic abscesses were the original cause of the pylephlebitis, for primary abscess of spleen is very rare, and the organ appeared quite hard and non-fluctuant till two or three days before the patient's death.

It seems almost certain that the patient's fatal illness was set up by the bristles found in the interior of the vein; though no trace of their passage could be found, they must after entering the alimentary canal have passed through the wall of the intestine and of the vein, and become lodged in the latter, possibly some time previously. Suppuration commencing around them, a general suppurative pylephlebitis was soon set up; the spleen would appear to have become greatly engorged with blood and enlarged from the thrombus in the splenic vein, and the organ ultimately became the seat of abscesses from the pyæmic blood condition.

According to Frerichs (*Diseases of the Liver*), there is but one other observation of traumatic pylephlebitis recorded in medical literature; it was that of a case published by Lambron in the *Archiv. gén. de Méd.*, 1842, p. 129, in which a fish-bone passed through the wall of the stomach and became impacted in the superior mesenteric vein, giving rise to a fatal suppurative pylephlebitis.

From time to time cases are recorded of splenic abscesses found *post mortem* which it is impossible to account for; it would be well in future in such cases to search for injury to the intestinal coats or veins from the possible presence of foreign bodies.

There are points of subsidiary interest worthy of note. The one is the hæmorrhagic nature of the fluid which was effused into the pleura; this seems to have been due to the cachectic condition of the patient, in part arising from disturbance of the functions of the spleen. In the case of splenic abscess recorded in this Society's 'Transactions' for 1894 by Dr. Finlay the patient coughed up several ounces of blood, though at the autopsy nothing abnormal was found in the lungs except some œdema.

The other point is that when the spleen suppurated, it became so softened that its outline was lost, and this gave the erroneous impression that the previous enlargement of the organ had subsided.

XXXVIII.—*A case of Thickened and Contracted Mesentery simulating Tumour in a case of Cirrhosis of the Liver.* By F. Lucas Benham, M.D. *Read May* 24, 1895.

G. R., a man æt. 55, married, with three grown-up children. By trade a carpenter, but has been disabled from working for the last two years by failure of health.

He has been for a very long time addicted to drinking to excess,—formerly spirits in large quantities, often a pint of gin in a day; more recently he has taken spirits chiefly in the form of "nips," and has drunk more beer. Patient's bodily and mental condition when I saw him prevented his giving answers to my questions, but his wife was aware of his habits of drinking to excess, and admitted the above facts.

He had an attack of jaundice fifteen years ago. Usually has retched in the morning, and never took any breakfast. Never had vomiting or hæmatemesis. Began to get rather thin and ill eleven months ago; his body swelled, but this swelling went away. He has been kept to bed for the last four or five months, but though he became weaker his friends did not notice that he continued to lose flesh. He has had but little medical treatment. Was admitted to Westminster Hospital two months ago with considerable dropsy, but refused to stay in there more than a week.

He was first seen by me on August 16, 1894. He was lying on his side in bed, looking emaciated, very cachectic, and helpless. Mind constantly wandering; could hardly understand or answer anything coherently. He is said to be more restless, talkative, and delirious at night. Tongue red and dry, sordes on lips. Slight cough, hawking and expectorating scanty blood-stained mucus. Breath offensive. Chest cannot easily be examined, but air apparently enters freely all over; no râles. Respiration not accelerated; heart normal; pulse regular, about 60. Skin cool, sallow but not jaundiced. Purpuric spots on extremities. Small bedsore on one shoulder. No visible enlargement of superficial abdominal veins; girth 39½ inches, abdomen being enormously distended. Fluctuation marked—dull nearly all over, in front and in both flanks; liver cannot be felt, or its area of dulness

well defined; it appears small; scrotum œdematous; very slight œdema of ankles; has a fanciful appetite, almost complete anorexia; takes very little food of any kind; is occasionally sick; bowels loose; urine scanty, high-coloured.

August 19.—He has remained much the same, but seems to get weaker and more helpless; the weight and encumbrance of his abdomen interfere with his moving in bed, and prevent his being able to raise himself at all, and cause some dyspnœa. He passes urine and fæces in bed. Lies half unconscious, but can be partially roused, and mutters when addressed. Girth 37½ inches, *i.e.* 2 inches less. Is said to have slept better last night; pulse and tongue as before; no cough. It was decided to perform paracentesis, merely to give relief, for it was plain that his life could not be prolonged for many days. It was attempted to make him sit up, but from his weakness and the protuberance of his abdomen this was impossible. He was therefore placed on the side of the bed with his legs over the edge, and his back supported in the semi-recumbent position with pillows.

A plain trocar and cannula were inserted midway between the umbilicus and pubes, but failed to draw off any fluid; probably the instrument did not enter the cavity. An aspirator was then used instead, and fluid at once escaped freely. It was clear reddish serum, and flowed continuously. Thirteen quarts were removed, and the abdomen was even then by no means empty. When the fluid had been partly evacuated, considerable pulsation was noticed in the epigastrium, evidently communicated from the abdominal aorta; and as the abdomen became more empty it assumed a peculiar form in front, a prominence remaining between the ensiform cartilage and the umbilicus, while the skin around was flattening and becoming depressed. On palpation a large irregular tumour, hard in consistence, of considerable area and thickness, was felt. It was uneven or coarsely nodular on the surface and margins, which were roughly circular in shape and tolerably well defined, slightly movable, and painful and tender on handling. Part of its anterior surface seemed to stand out in the form of a large sausage. It was situated deeply, reaching from the spine to the anterior abdominal wall, was longer from above down than from side to side, and projected further to the right than to the left of the middle line. It seemed to be distinct from the liver, but the lower border of the latter could not well be made out.

August 20.—Has been easier; can turn about; slept well;

less delirious, but still mind and attention are not clear; takes food a little better; passed more urine.

August 21.—Took no food since he was visited yesterday; slept nearly all the day and night, and died quietly this morning..

Post-mortem (seven hours after death).—Abdomen only was opened. Trunk fairly nourished; abdomen much swollen; face and limbs shrunken; much hypostatic congestion, especially of left arm; rigor mortis commenced; subcutaneous fat abundant. On opening peritoneum a large quantity (about 1½ galls. ?) of fluid escaped, similar to that removed by paracentesis. It contained no lymph or pus. When the fluid had escaped, the intestines were seen to be universally of a dingy brown or black colour; the parietal peritoneum was only discoloured here and there. The coils of intestine were nowhere adherent, but were huddled up together, and seemed firmer and more substantial than usual. Their external surface, besides being dark-coloured, had lost its smoothness and gloss, but there was no exudation of lymph whatever.

Liver.—Much contracted and flattened, coarsely granular (hobnailed); margins irregular. The capsule could not possibly be stripped off, though it was not much thickened and not adherent to surrounding parts. On section the organ was extremely dense, tough, and indurated. Structure—bright yellow lobules embedded in variable proportion in a quantity of the densest fibrous tissue; in places the glandular tissue was quite gone.

Spleen.—Large, capsule not thickened; consistence firm.

Pancreas.—Dense and indurated.

Kidneys.—Quite normal, apparently, in size, structure, and consistence; capsule very thin and stripped easily; surface smooth; organs venously congested.

Mesentery.—Remarkably shrunken and condensed, shortened and thickened in all parts; contained a thick stratum of fat between its layers, so that it formed a mass of very firm plates and rolls, drawing the coils of intestine together and up towards the spine. On the surface lay the thickened omentum in a cylindrical form. It was evidently this extreme condensation of the mesentery, mesocolon, and omentum that produced the mass by which pulsation was transmitted to the abdominal surface during tapping, and was taken for a tumour when the fluid was removed. The thickening of the mesentery involved its whole length and breath. The peritoneal covering of the

gut seemed also thickened, so that the tube was firmer and less collapsible than usual.

Remarks.—I have ventured to bring forward this case of a disease so common as cirrhosis of the liver, as I believe it presents certain features and complications which are worth notice, as they are somewhat unusual, and have not received much attention hitherto, as far as I know.

I am referring to the condition of the mesentery. In most of the text-books this condition is not mentioned at all, though it is well described in Hilton Fagge's book as the commonest lesion of chronic peritonitis. He states that its causation is not well understood, though it is usually associated with chronic Bright's disease or perihepatitis, neither of which was present in my case; and he makes no mention of its occurrence in conjunction with cirrhosis of the liver. If peritonitis occurs in the course of cirrhosis of the liver, it is usually of the exudative form, and is, I believe, generally traumatic, as a result of tapping.

It is common, however, to get slight forms of contraction of the mesentery in cirrhosis, at any rate affecting the omentum only.

I should like to raise the question whether this change in the mesentery is essentially a chronic peritonitis, or whether it is produced by a process of cirrhosis due to the same causes which bring about cirrhosis of the liver.

I have left a practical point till the last, viz. the existence of the tumour. As this is, of course, not perceptible until the ascitic fluid has been removed, it is easy to understand how such a thing may have escaped recognition, and may generally continue to do so, as the binder that is used prevents thorough examination. But if it is noticed, it may forcibly suggest a doubt, such as I felt in this case for the time, whether the tumour may not be of a malignant nature, thus making one alter one's diagnosis, and lead to an error in filling up the certificate of death if no *post-mortem* examination is made.

XXXIX.—*A case diagnosed as Tubercular Meningitis in which recovery took place, a brother aged nine months having died in the meanwhile with similar symptoms, and Tubercular Meningitis having been found on post-mortem examination.* By Samuel West, M.D. *Read May* 24, 1895.

Rhoda A., æt. 2 years and 7 months, was admitted under my care into the Royal Free Hospital for frequent vomiting, loss of appetite, and constipation.

She had been quite well till April 3, when she became feverish and vomited frequently. These symptoms continued until the 5th, when she had a fit. Her mother stated that she became stiff, was convulsed, and her eyes rolled. She remained in this condition for two days, when she seemed to rally a little, but she hardly slept at all, picked her face a good deal, and seemed to dread being touched.

On the 11th she was brought to the hospital and was admitted.

The patient was a well-developed child. She lay on the left side with the head drawn back, and seemed to take little notice of her surroundings. The eyebrows were slightly contracted, the eyes half open, the pupils and the movements of the eyes normal.

She was continually grinding her teeth. Except for a little bronchitis, there was nothing to note on examination. The pulse was rapid (165), and the temperature rose to 103° in the evening.

The next day, April 12, the child was very drowsy. On the 14th a patch of broncho-pneumonia was found at the right apex.

April 17.—Patient was worse, more restless and irritable, and head more retracted. The temperature, which till the 16th had oscillated widely, reaching 103° every evening, was now lower, not rising above 102.

April 18.—Very delirious at night; vomited twice.

April 20.—Seemed worse; slight squint observed for the first time; pulse irregular, but slower (120).

April 23.—Temperature had been subnormal for the last

few days, but the pulse was still rapid, 160. The right pupil was smaller than the left, and the squint continued.

April 27.—During the last few days the child had seemed better, though the temperature had again risen daily to 101° or a little more. The vomiting had continued about once a day. The squint was present, but not constant; both pupils were dilated, but the left most. The chest signs had completely disappeared.

April 28.—I examined the eyes and found double optic neuritis, most marked in the left eye.

May 2.—Patient was semi-comatose, vomited constantly, and had to be nasal-fed.

May 3.—The pupils scarcely reacted at all to light, and the child was much worse.

May 4.—The breathing became very irregular, two long deep inspirations being taken, followed by a variable number of short ones at long intervals. Respirations only about eight to the minute, pulse 154, temperature subnormal. During the night the child had an attack of twitching all over. At this time the child seemed to be dying, and of what seemed to be, without doubt, tubercular meningitis; but from this time she began to improve, though but slowly for a long time and with relapses.

On the 15th she began to take a little notice; the squint continued, though to varying degrees at different times.

On the 17th she lay upon her back for the first time for six weeks.

May 25.—Patient cried a good deal, as if from pain in the head.

On the 28th the following note was made by me:—"The twitchings and movements in the arms, which have been ever present since first noticed on the 4th, still continue, though they are now less severe. The twitchings are constant though varying in degree, affect the whole body more or less, and are well marked in the extremities and face. At times the legs and arms become quite stiff, and remain so for some minutes."

On the 30th the retraction of the head was greatly increased and the spine arched, but the twitchings were not quite so marked.

During the next month, though the symptoms varied from day to day, there was on the whole an improvement. The temperature had been normal, and the pulse and respirations only slightly accelerated. She ate and slept well, but had crying fits at night, when she made a great noise. The

retraction of the head and stiffness of the legs continued. The knee-jerks were absent, as they had been for some time. The eyes reacted to light. The child answered now when spoken to, and smiled.

July 18.—The optic neuritis had almost entirely disappeared, but the child seemed hardly able to see, though the pupils reacted to light.

On September 6 the child had a bad relapse. She screamed a good deal at night, lay with the head strongly retracted and the back arched, and took no notice of anything. In a few days these symptoms subsided again, and by the 19th she was much better and brighter. She had lost the retraction and arching, answered readily when spoken to, and seemed to be able to see a little.

By October 26 she had made much progress, now understood well what was said to her, saw better, and cried but little at night.

By November 1 she was able to sit up, and could speak a little.

By December 1 she was able to walk with a little assistance, and saw well.

On December 15 I examined the eyes and found the discs had almost completely recovered.

At the present time, February 8, she is running about actively; she sees perfectly, even to pick up very small things; she is constantly chattering, and speaks plainly. She appears now to be in excellent health.

One further fact of great interest remains to be stated, viz. that during this child's illness on June 19 her little brother, aged nine months, was admitted into the hospital under the care of Dr. Sainsbury, with a history of having been ill for three weeks with vomiting and fits. On admission there was marked retraction of the head, tense fontanelle, varying squint, and frequent vomiting. The discs were normal and the temperature not raised.

The child was in the hospital seven weeks, his condition fluctuating from day to day, but steadily deteriorating. The temperature began to rise on August 12, and the child died on the 16th. The diagnosis had been made of tubercular meningitis, and on the autopsy that lesion was found, but the tubercles were not very numerous nor the amount of inflammation great, though both were distinct enough.

The pathological condition is interesting, because the infant did not die until ten weeks after the commencement of his ill-

ness, from which it might be presumed that the inflammation was not very severe or extensive.

There is no reason *a priori* why recovery should not occur sometimes with tubercle in the meninges, as it does frequently with tubercle in other parts of the body, but it will be admitted that instances of it are very rare. The whole question turns upon the correctness of the diagnosis, and this is often a matter of opinion and judgment, for conclusive demonstration fails in the absence of an autopsy. Basal meningitis from other causes than tubercle is, no doubt, often recovered from. The whole question turns, in a case of recovery from presumed tubercular meningitis, upon the evidence of its having been due to tubercle. This evidence in many cases is not strong. Yet, considering the fact that in the great majority of cases of tubercular meningitis the correctness of the diagnosis is proved by the post-mortem examination, the diagnosis is not really as difficult as it might seem. Now the special interest of the present case lies in the fact that two children of the same family were attacked with similar symptoms at about the same time, the same diagnosis was made in each case, the one patient died, and the diagnosis was confirmed by the autopsy. The presumption is, therefore, that the diagnosis was correct also in the other patient who recovered. Moreover both cases ran a somewhat unusual course, for in the fatal case the duration was long, and the symptoms pointed to the absence of any considerable inflammatory exudation, a presumption also confirmed by the autopsy. It is just in such cases that recovery might occur, for the fatal result in most cases is due to the pressure on the nerve-centres by the inflammatory effusion.

While fully recognising the difficulties of diagnosis, I believe that the case I record was one of tubercular meningitis, a diagnosis which I made at the first in the early part of the case; and though the unexpected recovery of the patient has compelled me to review the facts very carefully, I am still of the same opinion.

Note.—For the use of those interested I append a few references to some of the recent literature of the subject:

Bonchut.—*Paris médic.*, 1882, vii, 241.
Carter.—*Med. Times and Gazette*, 1881, i, 378.

CHEADLE.—*Brit. Med. Journ.*, 1879, ii, 986.
DUDON.—*Mém. et Bull. Soc. méd. et chir. de Bordeaux*, 1876, 95.
DUJARDIN-BEAUMETZ.—*Bull. et Mém. Soc. méd. d'Hôp. de Paris*, 1879, 2nd s., xv, 256.
FONCART.—*Fr. méd.*, 1879, xxvi, 466.
GISCARO.—*Rev. méd. de Toulouse*, 1879, xiii, 37.
LEARED.—*Med. Times and Gazette*, 1864, i, 121.
MARTEL.—*Rev. internat. d. Sc. méd.*, 1886, iii, 287.
REIBEL.—*Soc. de Méd. de Strasburg*, 1889, xiv, 32.
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XL.—*A case of Calculus of the Kidney associated with a Papilloma in the renal pelvis: nephrotomy: nephrectomy: recovery.* By William Henry Battle. *Read May* 24, 1895.

A MAN æt. 51, an engine-fitter, was admitted into St. Thomas's Hospital on January 2, 1893, under the care of Mr. W. Anderson, for hæmaturia.

Six years before admission he had passed a large calculus and several smaller ones *per urethram*, but for some years before that had had severe pain at times of a cutting character in the left loin, and radiating round into the bladder; this pain was increased by exertion. The urine was then unchanged in appearance, excepting for three days after the passage of the calculi, when he saw blood.

In April, 1892, he had blood in the urine for two months; in May he passed some more calculi the size of small shot, and states that he was unconscious for three weeks. The bleeding stopped, recommenced in the middle of July, and continued on and off with intervals up to admission. He had passed two more calculi a week before he came in.

On admission the urine was smoky from the presence of blood, but contained no pus, casts, or crystals.

On January 7 the bladder was sounded with negative result, and on the 21st it was examined through a suprapubic incision. There was no growth or stone in the bladder, but blood was seen issuing from the left ureter.

In consequence of the absence of Mr. Anderson the man came under my care early in February, and I examined the abdomen thoroughly when he was under ether, but was unable to detect any enlargement of kidney or growth in the abdomen. It was not easy to absolutely satisfy oneself as to the exact condition of the abdomen because the man was rather fat and the abdomen large, with well-developed muscles. As a result of this examination the quantity of blood in the urine was increased for a few hours. Chemical examination of the calculi passed in December showed them to consist of oxalate of lime with some organic matter.

On February 23 lumbar nephrolithotomy was performed by me, and several dark-coloured stones of rounded outline

were removed; none were large, but the smaller sizes were very numerous, many being no larger than grains of sand. The pelvis of the kidney was explored by the finger for suspected new growth, and in the lower part of the anterior aspect a curious soft moss-like villous tumour was found, covering a base of about 1½ inches in extent; there was no hardness whatever about it, nor did it resemble in any manner a malignant neoplasm. There seemed, therefore, no reason why a local removal of the growth should not be attempted, and it was thoroughly scraped away with a sharp spoon, some of the kidney substance being brought away at the same time. The growth removed was very like a collection of small red mud-worms to the feel, and consisted of minute branched interlacing filaments which floated out in water. A drainage-tube was placed in the pelvis of the kidney, and the wound closed in the usual way. The shock resulting from the operation was slight. The growth was pronounced by the pathologists to be squamous carcinoma.

Next day (24th) the urine contained much blood, and the evening temperature reached 100°. On the 25th the evening temperature was 102·2°. There is no other record worthy of note.

On March 6 a trace of blood was found on chemical examination.

On the 16th there was very little discharge from the wound, and he left the hospital on April 5 with the wound quite healed, and fully relieved of his symptoms.

He was told to return if the bleeding recurred.

The man was readmitted to the hospital on July 19, 1894. He stated that he had resumed work as an engine-fitter on June 25 of the previous year; in August there had been some return of the hæmaturia—very slight at first, and only at long intervals; it had become constant in November, and by Christmas was very severe. He was under medical care, and was obliged to keep his bed, but there was no improvement, and from Christmas until admission to the hospital he had been quite unable to do anything. This constant loss of blood was not accompanied by pain. He had passed two or three calculi twelve months ago, and one or two small ones since. On account of the constant loss of blood he had applied for admission six months ago, but was refused through a misunderstanding. Since Christmas he had been confined to bed almost entirely.

He was now very anæmic, but had not lost flesh. The left

kidney could be felt, was evidently slightly enlarged, and was adherent to the scar of the former operation. He complained of a little pain on manipulation. The temperature was normal, and there were no signs of disease elsewhere nor evidences of secondary growth. The urine, which was of a deep purple colour, measured 41 oz. during the first twenty-four hours after he came in, and was of sp. gr. 1012. A sediment examined microscopically showed a large number of pus cells (a new feature in the case) and blood-corpuscles, with one or two epithelial cells of uncertain origin. The urine yielded on boiling about two thirds albumen. On August 9 the renal swelling was decidedly smaller, and the man did not complain of pain on manipulation. There was less blood in the urine, but about the same amount of pus. The only medicine which appeared to check the loss of blood was hamamelis, and this but to a limited extent; the urine became smoky instead of red. His general condition improved under treatment, the evening temperature being, however, at times 99° or 99·8°. On August 15 nephrectomy was performed through the scar in the lumbar region. The cicatricial tissue was extremely dense. The kidney was adherent to the cicatrix and on separating the adhesions a small abscess was opened which contained about an ounce and a half of pus, and was continuous with the interior of the pelvis of the kidney, which was found to be enlarged. Two or three small stones were found in the abscess. The lower part of the kidney was difficult to separate, the adhesions being very dense, and in order to get at them properly the wound was enlarged, an incision being made downwards from the posterior extremity of and at right angles to the transverse one. The ureter was exposed and divided between two ligatures. The pedicle was ligatured in two halves with silk. The capsule was then stitched across the stump with three or four sutures of silk. The wound was washed out with 1 in 2000 perchloride of mercury solution, a drainage-tube put in, and cyanide dressings applied. The amount of shock resulting from the operation was not great, but in consequence of the man's previously anæmic condition it was considered advisable to bandage the limbs with flannel and apply hot-water bottles; the temperature did not fall. The next day he had passed 16 oz. of urine but slightly blood-stained, and there had been very little discharge from the tube. On the 17th he had passed 24 oz. of urine of sp. gr. 1012, without albumen, and no trace of blood or pus was

found on microscopical examination. The tube was removed at an early date, and the wound was practically healed by the 28th. There had been a small abscess in part of the wound, but the suppuration was limited, and did not retard progress to any marked extent, but caused a rise of temperature of two degrees in the evening for three days. The average daily amount of urine passed during the last fortnight of his stay in the hospital was 38 oz. He left for a convalescent home on September 19, and when seen ten weeks later had gained strength considerably, and was much less anæmic.

The history of this case, when he came under my care, made it quite evident that the patient was suffering from renal calculus, for in addition to the attacks of renal colic, which had always been on the left side, he had passed calculi by the urethra. The continuous and severe hæmaturia caused me to think that he was also suffering from new growth of the kidney, and taken in conjunction with the man's age pointed to epithelioma. The absence of enlargement of the kidney when this symptom of hæmaturia had been present so many months made it unlikely that the hæmorrhage was due to sarcoma. Continuous hæmorrhage as a symptom of calculus is very rare indeed, but I have recently removed a calculus from the pelvis of a kidney, the only evidence of the presence of which was continuous hæmaturia of some months' duration without pain; but such cases are exceptional. The characters of the growth removed at the first operation were those of an innocent one, and although I was not aware of any case in which the local removal of a growth like this from the renal pelvis had been attempted, it appeared right to me in this instance to try and save the kidney. Had it been possible to treat the man at an earlier date, when the base of the growth was less extensive, a thorough scraping such as that practised might have been successfully carried out. There are few cases of papilloma of the pelvis of the kidney on record even in pathological works, and I have since been able to find but one description of a similar growth for which operation has been performed. The question as to the cause of the growth can hardly be answered; it is scarcely sufficient to ascribe it to irritation of the calculi, for the number of patients who have been submitted to operation for renal calculus, of varied shape, number, and size, is very large; but association of calculus with papilloma is, as I have said, extremely rare.

At the time of the man's second admission there was a distinct change; it was probable that the return of hæmaturia to such a marked extent indicated renewed growth at the seat of the former one, but now a tumour was felt in the position of the kidney, and there was pus in the urine. The enlargement made it possible that this return of disease was considerable, but it was less than could have been expected after the known existence of carcinomatous growth of more than eighteen months' duration. The presence of pus in the urine, the tenderness on pressure, in a few days the diminution of size of the swelling which was observed, the absence of emaciation or evidence of secondary growth, made the prognosis more favorable than would have been implied from the report of the microscopical examination. It was, however, necessary to remove the kidney in order to arrest the constant hæmorrhage, which was now most serious in its effects, whether the disease should eventually prove benign or malignant. This was done as I have described, and a reconsideration of the microscopical preparations has enabled me to give a much more favorable prognosis to the patient than could otherwise have been the case.

CLINICAL CASES

DESCRIBED BY CARD.

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I.—*A case of Charcot's Disease.* By A. WILLETT. *Exhibited November* 9, 1894.

JOSEPH H., æt. 44, farm labourer. Six years ago sudden pain in the night down the back of the right thigh. In the morning the back of the thigh was black and painful, but patient could walk. No history of injury. Noticed swelling at outer side of right hip shortly after. Treated in King's College Hospital for two months—probable hip-disease diagnosed—painted with iodine. Since then swelling has gradually increased and limb has become of less use, with more pain, until two months ago, since which time the swelling has increased more rapidly. The pain is paroxysmal, worse at night after exertion during day, sharp and stabbing in character, situated in region of great trochanter and front of tibia. Has not lost flesh. Sickness in morning for three years.

No history of syphilis. Gonorrhœa 26 years ago. No previous illnesses of importance.

Family history.—No history of phthisis. One brother had "hip-disease" in childhood.

Present condition (October 26, 1894).—Well-developed, healthy-looking man.

Chest: some emphysema. Abdomen flaccid. No enlargement of liver. Enlarged right inguinal and (?) iliac glands.

On right side is a large pyriform swelling, extending from

iliac crest and tapering to a wasted thigh. Swelling is smooth and even in front, and dilated veins are visible. Behind it is most prominent over the outer part of the gluteal region, where it is red, slightly œdematous, and fluctuation can be plainly felt. In front it is densely hard except at outer part, where fluctuation is obtainable. In position of tensor fasciæ femoris is a *movable mass* of bony hardness. Above this the great trochanter can be felt. A *second* hard mass is felt just below iliac crest. A *third* mass can be felt on pubic portion of os innominatum.

Measurements:

Anterior superior spine to adductor tubercle	R. = 15½″; L. = 17¼″.
Small circumference of thigh	R. = 11″; L. = 12″.
Circumference of calf	R. = 10½″; L. = 11½″.
Circumference of thigh 5″ above patella	R. = 13½″; L. = 14¾″.
Greatest circumference round growth and perineum	27¾″.
Corresponding measurement on left	21½″.

By traction, length from anterior superior spine to adductor tubercle can be increased about an inch.

Right thigh is generally held in position of adduction with slight external rotation.

Active flexion through 60°; passive flexion to 90°. Grating felt in region of great trochanter on movements of limb. No pain on manipulation. Rotation and abduction and adduction much limited. Electrical reactions of muscles of legs and thighs normal. Knee-jerks absent. No Argyll-Robertson pupil. Bladder and rectum appear to act normally (but micturition is frequent when he catches cold). Urine: no albumen or sugar, faintly alkaline on admission.

November 5, 1894.—Aspiration; ʒij of blood-stained, slightly turbid, slightly viscid fluid drawn off. Microscopically: blood-corpuscles.

A quantity of fluid afterwards oozed out from puncture made by the exploring needle, and the swelling is now much less tense.

II.—*A case of Charcot's Disease of the Hip with Dislocation.* By ANTHONY A. BOWLBY. *Exhibited November* 9, 1894.

THE patient is a man æt. 58. His history is that he slipped and dislocated his hip ten years ago, the dislocation being produced with hardly any violence. Since that time he has walked very lame.

At present the affected limb is much shorter than its fellow. The upper end of the femur can be plainly felt on the dorsum ilii. There is evidently no head to the femur, but there is a great deal of new bone in the neighbourhood of the neck. The femur has great mobility on the ilium, and in walking moves up and down more than three inches. There is no pain whatever in the joint.

The evidence of tabes are as follows:—Lightning pains in limbs; dim vision; absence of knee-jerks; inability to stand with the eyes shut; pupils do not react to light.

III.—*Case of Pseudo-bulbar Paralysis.* By F. W. MOTT, M.D. *Exhibited March* 22, 1895.

R. W., æt. 55, followed the occupation of ship's carpenter as a young man, but after falling from a mast and injuring his head he gave this up. At thirty-five he had fits, melancholia, and paralysis of the left arm and leg, with weakness of the right side. He gradually recovered from this condition, but four years ago he found himself getting weak in the legs, and he had difficulty of swallowing and speaking. He attended Dr. Bond at the Throat Hospital, Golden Square, who was good enough to send him to me two years ago, and since then he has been under my care as an out-patient. When first I saw him he had marked difficulty in swallowing, and the saliva ran out of the angles of his mouth. Now, although he is feebler in his walk, which is very much like that of a man with paralysis agitans, yet his bulbar symptoms are better.

At the present time he has exaggerated knee-jerks, ankle-

clonus, and wrist-tap contraction on both sides, but no marked wasting of muscles or reaction of degeneration—an indication that *his pyramidal tracts are sclerosed,* but that it is not amyotrophic lateral sclerosis with bulbar symptoms. Moreover the bulbar symptoms are not like those ordinarily met with. The tongue is somewhat wasted, the soft palate does not move properly, the vocal cords cannot be seen, because with each inspiration the sides of the epiglottis fall in; doubtless the vocal cords are partially paralysed. The orbicularis oris is not paralysed. His speech is peculiar, it being a deep monotone, each syllable being uttered with great difficulty, and he forms his consonants mostly by protruding and pursing up the lips.

Although there is no distinct evidence of a *double* lesion in this case, yet the slow progress of the case, the improvement of the bulbar symptoms, and the obvious signs that both pyramidal tracts are sclerosed, render warrantable the assumption of it being a pseudo-bulbar paralysis. His intellect is good, although his tottering walk and curious speech necessitate his carrying a letter with him to say that he is a teetotaler and a respectable man; for he was, on account of his walk and speech, once fined five shillings for being drunk and disorderly.

The patient is also of interest in having complete transposition of the viscera.

Dr. Mott attempted to explain the bulbar symptoms by bilateral sclerosis of the pyramidal tracts interfering with the nutrition of the glosso-labial laryngeal nucleus, which lies immediately behind the decussation of the pyramidal fibres. Vessels which supply this nucleus would have to pass through the sclerosed tissue, and the cells would suffer from impaired nutrition owing to thickening of the vessel walls. This would account for the partial but not progressive wasting of the tongue.

IV.—*Case of Syringomyelia with perforating ulcer.* By J. H. Morgan, and F. W. Mott, M.D. *Exhibited March* 22, 1895.

W. S., æt. 25, was admitted into Charing Cross Hospital for perforating ulcer of the right foot. There is nothing noteworthy in the family history except that he has a *brother with clubbed feet.*

When aged sixteen he suffered a chill from wet clothes. A week later he noticed that he was walking on the outer side of both feet. A little later he had a piece of bone removed from the left foot. At 17—18 he noticed weakness and wasting of both hands. The left leg was amputated seven years ago at Guy's Hospital for perforating ulcer.

His present condition corresponds in many respects with the curious clinical phenomena met with in *syringomyelia.* There is *general wasting of muscles,* notably of the *small intrinsic* muscles of the hands and foot. *None of the muscles respond to faradism,* but there is not reaction of degeneration with galvanic current. The right foot is in a condition of talipes equino-varus and pes cavus. There was a large chronic ulcer on the outer side of the plantar surface, 4 inches long and 2 inches broad; the base covered with unhealthy granulations, the edge uneven, and the discharge foul: no surrounding inflammation or tenderness. This has now healed with charcoal poultices and antiseptic dressings.

No sexual, bladder, or bowel trouble.

There is marked lateral curvature of the spine, which the patient says came on with the leg troubles.

On testing the muscles with the faradic current it was observed that the patient *felt no pain* in any part of his body, neither does he feel a pricking sensation when *a needle* is driven into the skin. In both cases he only *feels something touching him.* There is, therefore, *analgesia* all over the body, but *no tactile anæsthesia,* except over the right leg below the knee. With heat and cold there is apparently diminished sensibility up as high as the second dorsal vertebra, but his answers are not very reliable.

It is perfectly manifest, however, that he does not feel painful sensations, and there is, therefore, *sensory dissociation.* Charcot believes that a central *myelitis* may be followed by syringomyelia, and the history of chill rather suggests the

possibility of this. On the other hand, many undoubted cases of *central gliosis*, followed by excavation, begin in this manner. The *absence of pain* with the onset, the progressive wasting of muscles, especially the *intrinsic muscles of the hands and feet*, the scoliosis, the *sensory dissociation*, and the *perforating ulcer* all point to a central lesion of the grey matter of the spinal cord.

V.—*Case of Progressive Muscular Atrophy with Argyll-Robertson Pupils.* By JAMES TAYLOR, M.D. *Exhibited February* 8, 1895.

THE man had much wasting of the upper limbs, especially in the muscles about the shoulders on both sides. The trapezius was wasted, even in its upper part—the *ultimum moriens* of Duchenne,—unequally on the two sides. When he first came under observation there was much fibrillary twitching in the muscles, but this had now ceased. There was also the condition of the pupils which still persists, viz. a loss of reaction to light while there was a brisk reaction during accommodation. The knee-jerks were active, there was no ataxy, and no pains or other sensory disturbance. There was a history of a chancre twenty years ago.

VI.—*A case of Myopathy, facio-scapulo-humeral type.* By C. E. BEEVOR, M.D. *Exhibited March* 22, 1895.

FREDERICK JOHN S., æt. 27, noticed when fifteen years old that he began to have weakness of the legs in walking, with weakness and wasting of the arms and face, but the symptoms came on so gradually that he could not say which part of the body was first affected. When he was twenty years old his walk became waddling, and he began to have curvature of the spine, while two years later he could not walk without assistance.

He has always had good health; there is no nervous disease in his family, and he has one brother and two sisters healthy.

The following muscles on both sides are weak:—the frontalis, so that he cannot elevate the brow; the orbicularis palpebrarum, so that he cannot quite close the eyelids, and he is unable to prevent them being opened by a very slight pressure of the finger; the elevator of the upper lip, the depressor of the lower lip, the orbicularis oris, so that he cannot whistle; while the tongue is normal and the soft palate, zygomatics, and corrugator supercilii act well.

In the neck both sterno-mastoids are wasted and hardly contract on flexing the head forwards, but the omo-hyoids, which are exposed in their whole course, stand out, and on rotating the head to one side the omo-hyoid of that side contracts. Both trapezii, with the exception of the middle part, are absent, exposing the levator anguli scapulæ, which can be felt to contract on shrugging the shoulders, and also the splenius and complexus. The supra-spinati and infra-spinati are slightly hypertrophied, but act well; the deltoids are much wasted, and do not contract; the pectoralis major is wasted in both parts, especially the sternal. The latissimus dorsi and serratus magnus are both wasted, and do not contract.

The biceps, brachialis anticus and triceps are gone, while in the forearms all the muscles are present and act except the supinator longus, which is completely absent on both sides; in the hands the small muscles of the thumb and the first interossei are wanting.

The recti abdominis are very weak, and cannot be felt to contract in trying to bring the body forwards when lying back in a chair; in fact, he performs this movement by arching the abdomen convex forwards, and then allowing the thorax to fall forwards by gravity. While the erector spinæ acts well and can extend the spine, the pelvis is tilted as far forwards as possible both in sitting and standing, owing to the glutei being wasted and not acting; hence the extreme lordosis on standing, and which persists on sitting down. Flexion of the hip is present and fair, abductors of thigh are wasted and do not act, extensors of knee very much wasted and no power; the flexors of knee contract feebly. There is no wasting below the knee, except of the peronei, causing the foot to be in the position of varus. He can just walk holding on to the shoulders of an assistant, but he cannot get off a chair without much help. With the exception of the supra- and

infra-spinati there is no hypertrophy. There is no anæsthesia and no affection of the sphincters.

The symptoms which show that it is a myopathy—a primary disease of the muscles—and not a disease of the medulla or spinal cord are (1) the distribution of the affected muscles, and notably the involvement of the orbicularis oris without the tongue being affected, (2) the absence of fibrillar contractions, and (3) the absence of the reaction of degeneration to the galvanic current.

There are two points of interest with regard to the muscles moving the head. In flexing the head forwards forcibly against resistance the omo-hyoids can be seen to contract on both sides, and in turning the head to the right the omo-hyoid on that side contracts; the depression of the larynx can, however, be felt in normal individuals in flexing the head against resistance. In extending the head backwards the complexus on either side can be felt to contract, but not the splenius capitis, but the splenius of one side comes into play as soon as the head is rotated to that side, so that the splenius is apparently not an extensor of the head, but only a rotator.

VII.—*A case of a Bullet Wound of the left Parietal Lobe with few symptoms.* By W. H. A. Jacobson. *Exhibited November* 9, 1894.

THE patient, a fine, healthy marine, while skirmishing at Madena Creek, West Coast of Africa, February 23, 1894, was struck on the left side of his head by a bullet, which lodged in the lower part of the left parietal or the junction of this and the upper temporo-sphenoidal region. He became unconscious at once, but remembered a little later being accidentally dipped into the water as he was being taken into a boat. He also remembered being helped up—*i. e.* being able to walk—the side of the ship at Bathurst. He remained here three weeks, and two weeks later was brought to Plymouth. During this time he remembers his food, his visitors, &c.

It is somewhat difficult to make out accurately what was the patient's state during the earlier weeks. He had no

hemiplegia, hearing was perfect, there was no interference with smell, no diplopia or crossed amblyopia. But there seems no doubt that for ten weeks he was the subject of visual aphasia and alexia. Words heard were readily understood, words seen in print or writing conveyed no meaning to him until he had spelt them through. This was the case with the simplest words. He was also quite unable to read. This condition lasted for ten weeks. Headaches frequently recurred, especially severe when the wound was closed for a time.

The surgical part of the case is of less interest. The patient was invalided to Plymouth, and for some months the wound used to close and then reopen, pieces of bone and of his cork helmet coming away. On June 27 an attempt was made to extract the ball, but as some brain matter escaped, the attempt was desisted from. In October he was sent to me by Dr. Morley, R.N., F.R.C.S. There was then a sinus 2 inches above and 1½ inches behind the left external auditory meatus. A probe introduced passed through a jagged opening in the skull upwards and to the left for 3 inches. Save for a liability to headache and the sinus the patient's health seemed excellent. On October 19 a flap was turned down in the usual way, exposing a jagged opening in the skull. This was enlarged with Hoffman's forceps, exposing the granulating mouth of a sinus-like track in the dura mater, this membrane being quite healthy immediately around. A little deeper, two small irregular pieces of bone were removed. A catheter now gave the impression of passing along a small tunnel-like passage for about 2 inches, where it struck a solid body, apparently encapsuled. The sinus having been well dilated, the solid body, which proved to be the bullet, was loosened from its bed with sharp spoons and extracted with dressing forceps. It was embedded in a mass of granulation tissue. A fellow-bullet weighs 172 gr.; they are made of hammered iron and facetted. During its extraction there was a slight but very distinct escape of brain substance. His recovery was uneventful save for a rise of temperature on the sixth day. This was due to my having removed the drainage-tube too early—on the second day. I ought to have made more allowance for the large amount of granulation tissue which still remained. When the site of the tube was opened up about ℥iij of sweet pus escaped. The wound is now practically healed, the patient free from headaches, and in excellent health.

The case is of interest physiologically as it bears somewhat on our knowledge of the functions of the part of the brain formed by the adjacent lower left parietal and upper temporo-sphenoidal lobes. It is another instance in which lesions of that area have caused visual aphasia and alexia. It is of further interest, perhaps, from its bearing on the question of interference when a ball is deeply imbedded in a part of the brain which is not of the highest importance. I would call attention to its large size and shape, so different from those of modern projectiles, such as the Lee-Mitford. It is a fact that in some cases of bullet wounds of the hemispheres which have been published as recoveries without operative interference, evil results have followed later, either from softening, from effusion into the ventricles, or perhaps, as shown by Flourier in his experiments, by the ball which at first lodged in the upper part of a hemisphere gradually travelling down towards the basis cerebri. It may be reasonably suggested that with such a foreign body any of the above late evil results might easily have followed.

VIII.—*A case of Trephining for Traumatic Epilepsy.* By Henry T. Butlin. *Exhibited November* 9, 1894.

R. B., æt. 55, carpenter, admitted into St. Bartholomew's Hospital September 4, 1894.

He walked into the surgery, saying that he had fallen about 8 ft. and had struck the back of his head against the mantelpiece. He was a little light-headed, and had an abrasion of the occiput in the middle line, also bruises of the left elbow and about the first dorsal vertebra.

Was very restless after admission, sometimes dozing, often wanting to get up and go home.

September 8.—At 3.30 P.M. suddenly cried out, and was then convulsed. A second fit occured at 3.40, and five more fits before 5.30 P.M. At that hour the eighth fit occurred. The eyes were turned slowly to the left, then the head was turned in the same direction. Immediately afterwards the left upper extremity became rigid, and this was followed (in order) by rigidity of the left lower extremity, the right lower

extremity, and the right upper extremity. Then the left side of the face became convulsed, and convulsions spread to the limbs in the order in which they had become rigid. The fit lasted for about 30 seconds. When it had ceased, there was left hemiplegia, which passed off in about a minute, leaving the left side a little weaker than the right.

The fits were repeated in similar fashion until the seventeenth fit at 8.48 P.M., which only differed from that which has been described in its duration, which was 90 instead of 30 seconds.

When Mr. Butlin saw the patient during and after this fit, there was paresis of the left arm and side of the face, while the lower extremity appeared to be little, if at all, affected.

Between the fits the patient answered rationally questions addressed to him.

There was no outward sign of injury to the right side of the skull, and there was no history of previous fits.

Diagnosis.—Irritation and some pressure on the cortical substance of the right side of the brain, most intense in the motor areas of the face and arm.

Operation.—Trephining with large trephine over fissure of Rolando, rather below the middle. Beneath the dura mater was a little blood-clot, not larger than the thumb-nail, and quite thin. In the subarachnoid space beneath it, a collection of clear fluid, beneath and (probably) by which the surface of the brain was deeply depressed. In order to be sure that there was no more clot, a second piece of bone was trephined out above the first. Both pieces were placed in a solution of boracic acid (10 gr. to ℥j). The little clot was removed, the arachnoid membrane was punctured, the fluid allowed to run out, and a tiny piece of gutta-percha tissue was introduced to prevent the fluid from again collecting.

The dura mater was replaced, and the two pieces of bone were placed upon it, after the lower margin of the lower piece had been chipped away in order to ensure free drainage of the subarachnoid space.

The fits did not recur after the operation, but the patient complained of great headache, and was at times so restless and active that he had to be constantly watched by a male attendant.

September 11.—Œdema of scalp about wound.

September 22.—Drainage tissue removed.

September 26.—A little pus for the first time since the

operation squeezed out from the place where the tissue had been.

September 28.—Has had to be removed to a separate ward on account of the noise he made at night. Still slight suppuration.

Wound opened up for Mr. Butlin by Mr. Lockwood. Some spongy granulations at the lower notched margin of the lower piece of bone. Both pieces of bone firmly fixed, so that it required a decided effort to remove the lower. The upper was left *in situ*, and the granulation tissue scraped away. Pure carbolic acid applied. Lowest part of wound drained.

Wound healed quickly; but the patient showed no signs of improvement for many days. The services of the male attendant could not be dispensed with until the 22nd of October.

The patient was quite well many months after the operation. No recurrence of fits.

IX.—*A case of Syphilitic Cranial Necrosis: brain compression and frequent fits: trephining: recovery.* By Herbert W. Page. *Exhibited March* 22, 1895.

ON July 15, 1894, William J., æt. 41, was found by a policeman unconscious in the street, and was brought to St. Mary's Hospital.

On admission he had rapidly recurring epileptic fits for a period of two hours. It was noted that the fits generally began with twitching of the fingers of the left hand, then the left arm and left side of the face were involved, then the left leg and afterwards the right arm and leg; the convulsions quickly became general, and consciousness was lost. The left limbs were afterwards paralysed. The various phases of the fits followed each other with such rapidity that it was difficult to observe their onset and march, but I think it is certain that some of the seizures began in the left leg. The fits varied very greatly in severity and number. In some he did not entirely lose his senses, in some only were his head and eyes turned to the left, not after all of them were his limbs paralysed.

Twelve years ago he had syphilis. For the past three years he had suffered from pretty constant pain in the head, and his health had been failing. A year ago he had a single fit, and since that time he has had occasional twitchings of his muscles; he has frequently felt giddy, and been obliged to sit down. During these attacks, which were not "fits," he said, his left arm would twitch, then the left side of his face, and then both his legs. He complains of pain over the right parietal region, and there is tenderness on percussion two inches vertically above the pinna. During the next few days after his admission his fits increased in severity and frequency, and on the 20th it was noted that he seemed much weaker in himself. He had had five fits in the night. On the 21st the report was more unfavorable, and he had to be roused to answer questions. He said he was inclined to see double as the fits came on. He has had as many as three or four in the hour. On the 23rd the fits were almost continuous, and during them the sphincters were relaxed. In twelve hours of the night of July 23, twenty-three fits were noted. On July 24 he was transferred to my care with a view to operation, the provisional diagnosis of cerebral tumour having been made.

On admission to the surgical ward the notes record that he is unconscious and roused with much difficulty. There is incontinence of urine and fæces. He has a fit about every ten minutes, the longest interval being fifteen, the shortest five minutes. When about to have a fit his right leg becomes abducted and rigid, then almost simultaneonsly his left limbs and the left side of his face are involved in violent clonic movements, followed by rigidity. In the interval of the fits there is complete paralysis of the left limbs and left side of the face.

The conclusion to which I came myself was that we had to do in all probability with a syphilitic lesion rather than a circumscribed new growth. There was the history of syphilis and of long-continued pain in the head, there were the evidences of an old node on the occipital bone, there was no optic neuritis, the scalp was suspiciously pulpy, and the nature of the fits seemed to point to a large surface lesion causing pressure rather than a cortical tumour. At any rate it was clearly a case for operation, and this was undertaken on July 25. A flap of scalp having been turned down from the right parietal bone with the view of trephining over the Rolandic area, a small quantity of dirty inflammatory matter

was found between the scalp and the bone, and the bone itself was seen to be eroded in many places. An opening was made with an inch and a half trephine. The bone all round was soft and eroded, and diseased parts of it were removed with Hoffmann's forceps, until the resultant hole was roughly oval in shape and measured some 2½ by 1½ inches. Lying between the bone and dura mater was broken-down leathery gummatous yellow matter, which was peeled away from the whole area exposed and for some distance beneath the bone. It is certain that much of this stuff was left behind; but inasmuch as a very large hole had been already made in the skull, I deemed it wiser to desist from further removal of bone in order to get at it, and all the diseased portions of bone had, I believed, been taken away. The dura mater itself, although considerably depressed by the inflammatory exudation, looked healthy, and it was therefore not opened. Brain pulsation could be distinctly felt. The flap was closed in the usual way and without drainage.

The man seemed no worse for the operation, but for six days had a subnormal temperature. One hour after it he had a fit. During the night of the 25th he had twelve, and on the 26th several fits. On the night of the 26th he had two, and during the afternoon of the 27th one fit. On July 28 the wound was dressed and exit was given to some bloody serum from beneath the scalp. He was then perfectly conscious and free from pain. The wound healed without further interference. He soon regained the power of moving his arm and leg, and the facial palsy subsided. He was sent to a convalescent home on August 24, and on his return home in six weeks resumed work. He has had no fit since July 27, and has been free from pain. He is perfectly well at present—far better, he says, than he has been for years.

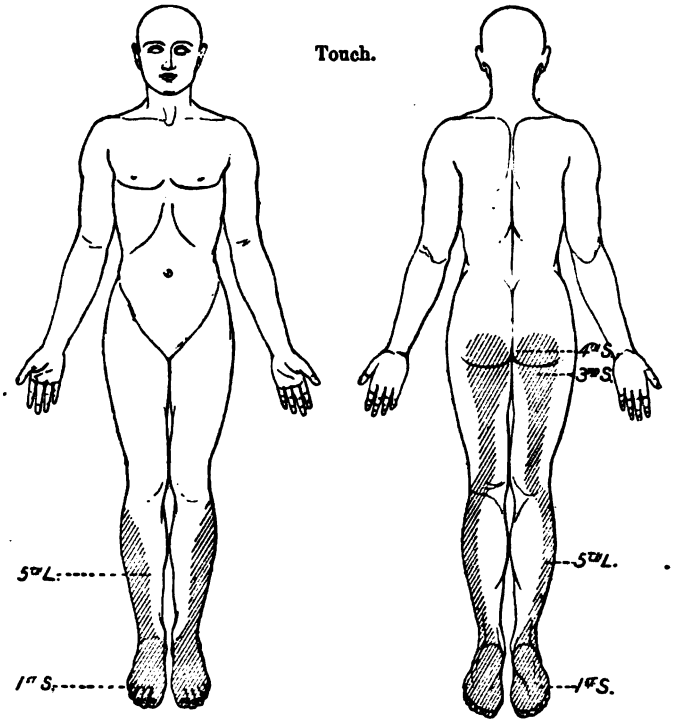
I presume that all this patient's symptoms were due to pressure on the brain, acting with greatest potency over the whole Rolandic region. And had it not been removed he must soon have died. In 1890 I had a case very like it under my care in St. Mary's, in which spontaneous relief from cerebral compression had been given by perforating erosion of the skull and protrusion of inflammatory matter through the scalp. In that instance also the condition was at first thought to be due to tumour, but it was wholly syphilitic. The man died of pyæmia after a long illness.

X.—*A case of Fracture Dislocation of the Spine after Operation.* By C. A. BALLANCE, M.S. *Exhibited March* 22, 1895.

ALBERT J., æt. 40, railway guard. Admitted November 10, 1893, into the National Hospital, Queen Square, under Dr. Bastian.

In June, 1893, he fell from a rapidly moving train. He did not lose consciousness, but found that he was unable to rise, as he could not move his legs. He was unable also to pass water. During the next few months his condition

FIG. 21.



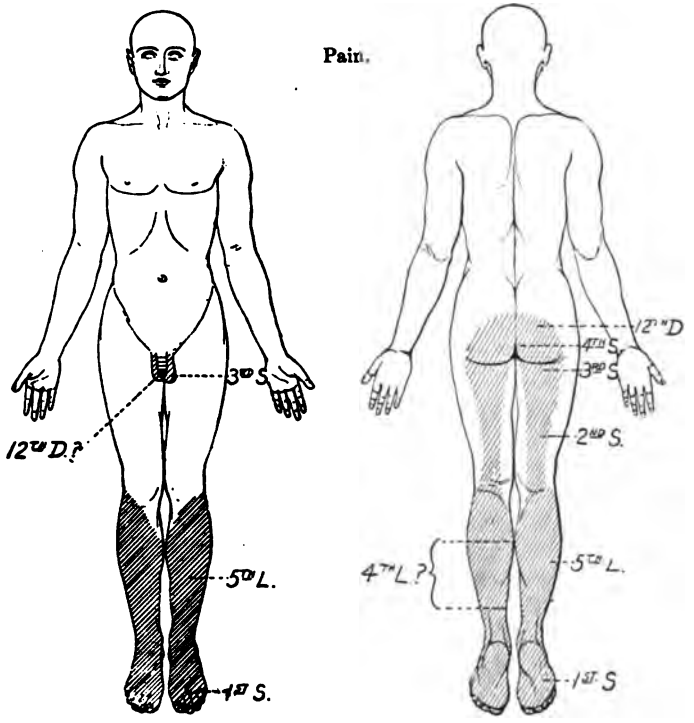
gradually grew worse, especially in consequence of his having cystitis and incontinence of urine and fæces.

Condition on admission (November 10, 1893).—Patient

complains of pains in the knees which prevent sleep, inability to sit or stand, loss of power in the lower limbs, and incontinence of urine and fæces.

Lower limbs—motion.—Ankles and toes—movements entirely lost. All muscles below knees paralysed and wasted. Knees—flexion almost absent, extension weak. Hips—adduction and flexion good; abduction or extension feeble;

FIG. 22.



wasting of glutei muscles and hamstrings: these muscles as well as those below the knee give the reaction of degeneration.

Sensation.—Touch: anæsthesia nowhere absolute. Pain, and heat and cold areas definite and with well-marked outlines (see Figs. 21, 22, and 23).*

* Sensation charts taken in November, 1893, before operation. Line shading = marked anæsthesia. Dot shading = relative anæsthesia.

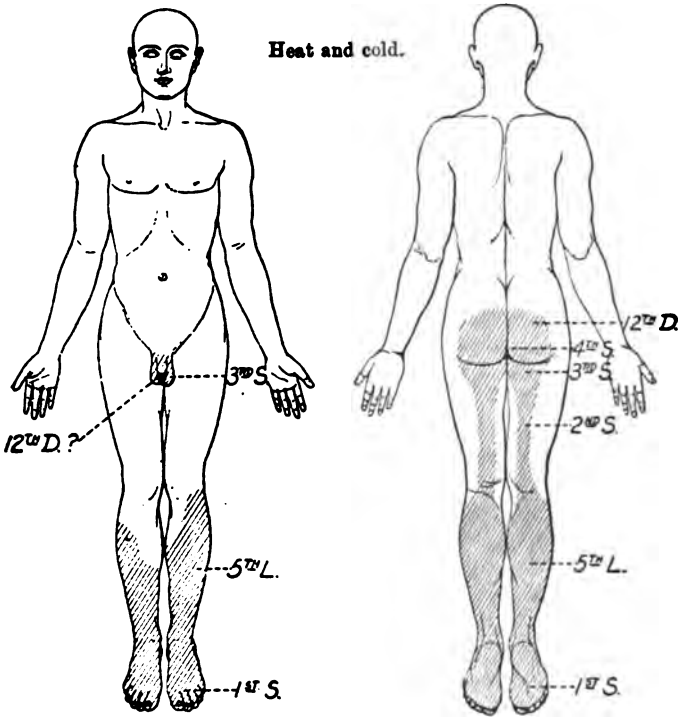
Knee-jerks with difficulty obtained, and diminished. Plantar reflexes absent. No ankle-clonus. Cremasteric and abdominal reflexes present.

Incontinence of fæces. Constant dribbling of offensive urine.

The twelfth dorsal spine projects markedly, but there is no tenderness about the spine in this region.

The condition of the patient remained unchanged until

FIG. 23.

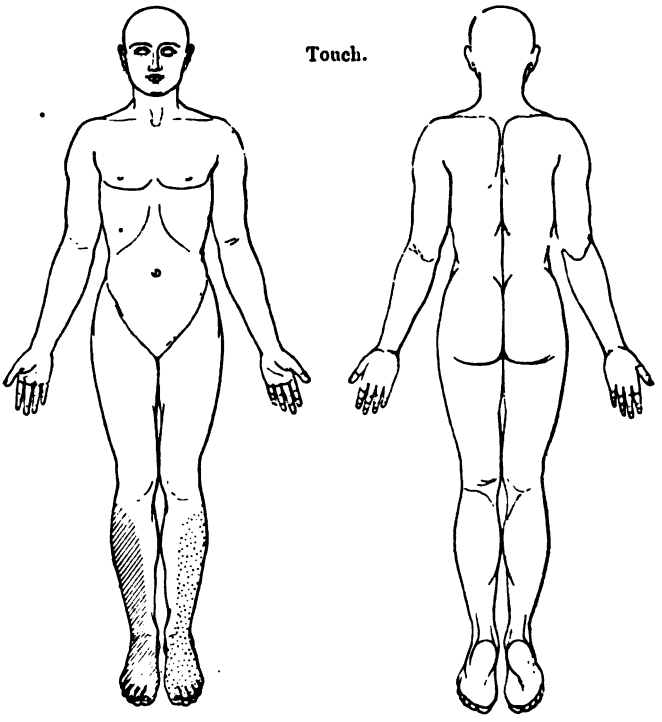


the day of operation, except that the cystitis improved under treatment. The urine, however, continued to constantly dribble away: the bladder was small, and was never distended except during the treatment by antiseptic irrigation.

Operation (January 12, 1894, seven months after the accident).—1. A vertical incision 7 inches long (the centre of which covered the projecting twelfth dorsal spine) was made.

2. By pushing aside the muscles the laminæ of the tenth, eleventh, and twelfth dorsal, and the first and second lumbar vertebræ were exposed. At this time there was much bleeding, in consequence no doubt of the new scar tissue around the site of injury: it was controlled by pressure.

FIG. 24.



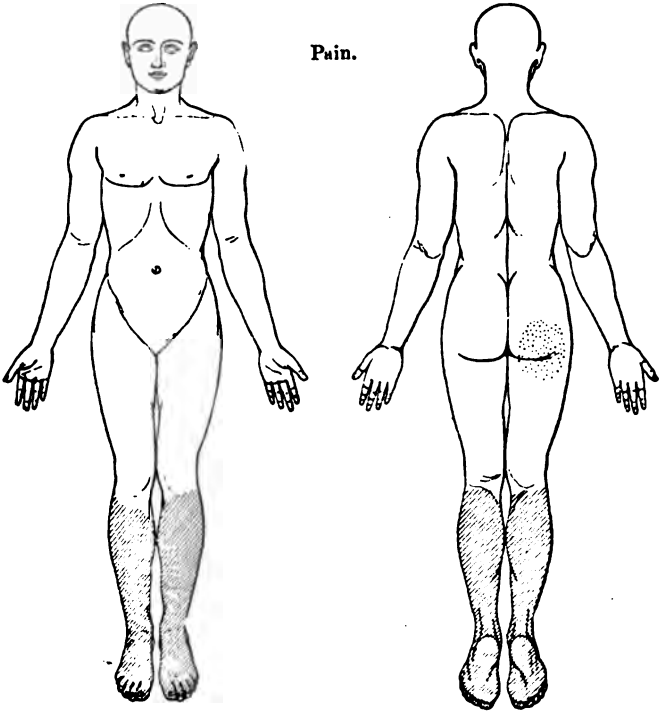
3. The spinous processes of the tenth, eleventh, and twelfth dorsal and the first and second lumbar vertebræ were now cut away.

4. When all the exposed laminæ had been thoroughly cleared it was apparent that the laminæ of the first lumbar vertebra were on a plane anterior to those of the twelfth dorsal vertebræ, there being a marked interval between the upper border of the former and the lower border of the latter.

5. With some difficulty, in consequence of the altered

relations of the parts and the presence of bars of new bone, the laminæ of the eleventh and twelfth dorsal and first and second lumbar vertebræ were taken away. The dural sheath was compressed by and adherent to the laminæ of the first lumbar vertebra, especially along their upper borders. From the spinal canal were removed some sharp spicules of bone and some scar tissue on either side of the theca opposite the twelfth dorsal and first lumbar vertebræ.

FIG. 25.



6. The theca was not opened, but was exposed for about 4½ inches. Above and below where the dural sheath was visible the spinal canal was quite free, as determined by the passage of a dura mater elevator.

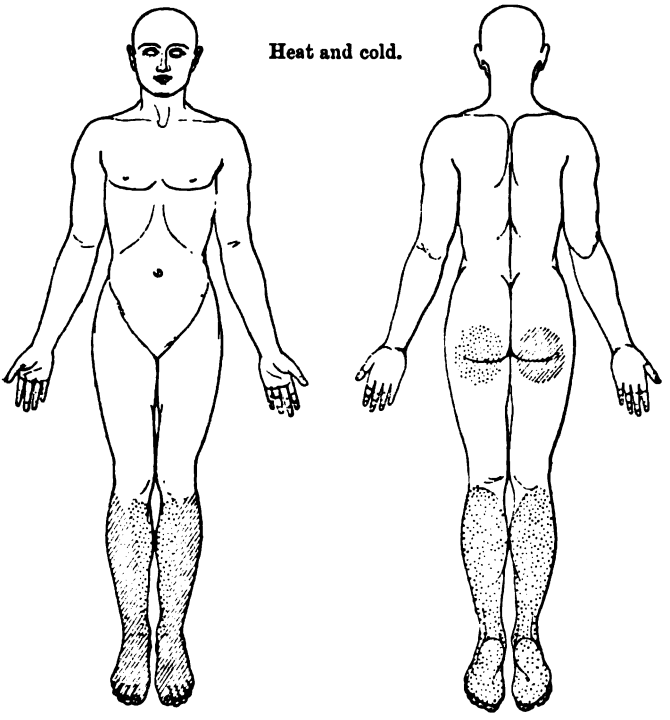
7. The wound was treated in the usual manner, and healed by first intention.

Two months later the following note was made:—March

19, 1894.—All movements of hips and knees good. Sensation—much improvement. Sphincters—some control; no cystitis.

Present condition (March, 1895).—*Motion.*—Ankles and toes—no movement. Hips and knees—all movements strong and normal.

FIG. 26.



Sensation.—See Figs. 21, 22, and 23.* Much improvement. Full control of rectum and bladder; no pains in the lower limbs; general health good; walks well in boots (made by Ernst) and crutches.

Remarks.—(*a*) It seemed doubtful that an operation so long a time as seven months after the injury would do much good. The patient, however, was losing ground, and it was his only hope; the complete paralysis of the bladder with dribbling was the most unfavorable sign, but it was difficult

* Sensation charts made in February, 1895, thirteen months after operation.

to think that the lower end of the cord was crushed, seeing that there was nowhere absolute loss of tactile sensation, though there were extensive skin areas with absolute loss of consciousness to sensations of pain and heat and cold. On the whole it seemed probable that the upper extremity of the cauda equina was compressed, and that the cord had escaped by an anatomical variation, namely, by not extending down as far as the first lumbar vertebra. The favorable result justifies the view taken before operation.

(*b*) At the operation I was in doubt as to whether to open the dural theca or not. With the theca open it might have been possible either to remove scar tissue which had taken the place of blood-clot, and which was acting injuriously by compressing nerves, or even to resect and suture a nerve-root. The theca, however, was not incised, for the reasons that there had been no increase of paralysis as far as I knew since the period of the accident, and that an intra-dural operation must of necessity entail more risk than one which is extra-dural.

(*c*) With regard to the spinal root segments involved, they were probably the twelfth dorsal, fourth and fifth lumbar, and the first, second, third, and fourth sacral. The least improvement as the result of the operation is manifested by the fourth and fifth lumbar and first sacral (see charts).

(*d*) The muscles below the knee-joints are completely paralysed. The man is practically in the state of a patient who has had amputation of both lower limbs below the knee-joints performed. The question arises, would this patient be improved by a double amputation at the ankle-joints or below the knee-joints?

(*e*) The case seems to offer a good illustration of the usefulness of operation (even after a long interval of time has elapsed since the injury) when the cauda equina is the part damaged and not the cord.

With some operations upon the central nervous system there is a loss account as well as a profit to be considered in rightly judging of their value. In this case we have only to reckon the gain, though I wish it had been still larger.

XI.—*A case of Local Asphyxia of One Hand.* By J. P. Parkinson, M.D. *Exhibited March* 22, 1895.

THIS patient, a domestic servant æt. 19, says she was strong and healthy till three years ago, when she began to notice aching pain in the left hand and fingers, accompanied by a reddish swelling of the whole dorsum of the hand and fingers; at this time there was no loss of sensation. This condition lasted two months, then the swelling abated for a fortnight, but returned on her resuming work, and this and the pain continued on and off till she permanently left off work in November last year. At this time she was operated upon for a ganglion at the base of the flexor tendon of the middle finger, but this appeared to have no effect upon the condition of the back of the hand. In December last year swelling and pain returned in the same situation as before, and since then whenever she attempted to use the hand this result has followed. In December the back of the hand and fingers were noted to be bluer than usual, and on exposure to cold turned almost black, while sensation of all kinds was *lost on the inner side of the back of the hand and fingers*, and a certain amount of *swelling appeared on the dorsum* of the hand, also, occasionally, *black patches* about the size of a shilling appeared on the back of the hand, which did not spread, but gradually scaled off, leaving natural skin beneath.

There is no history of rheumatism or ague. Menstruation ceased four months ago, but returned on March 18.

The condition now is less marked than I have ever seen it before, for a week ago the back of the hand was dark blue in colour, and the hand felt distinctly cold. On pressure the spot becomes dead white, but soon a vermilion-coloured patch appears, which extends for a time and then fades gradually.

The skin shows signs of previous swelling, and the nail of the little finger shows a transverse groove, but there is no clubbing, nor striation of nails.

At times she has severe pricking sensations in the hand and ring and little fingers.

Pressure on the nerves at the elbow causes no shooting down the forearm, as it does on the right side. The urine has never been brown, and examination shows no abnormality.

The cardiac system appears normal, and there is no difference in volume of the pulses.

Points of interest in this case are the unilateral distribution of the affection, and that most of the symptoms are referred to the distribution of the ulnar nerve; the appearance of the hand now suggests the beginning of a process of scleroderma.

XII.—*Meningocele in region of Lateral Fontanelle, probably traumatic.* By H. Betham Robinson. *Exhibited March* 22, 1895.

ARTHUR G., a child æt. 7 months, first came under my care in December last with a swelling on the side of the head over the posterior part of the parietal bone. The history of the case is as follows:—The child is the first-born of a healthy-looking mother, and was born after some difficulty. The presentation was cranial, and instruments were used. The head appeared to be bruised all over, but most of this swelling went away within the month, leaving the tumour which now concerns us. The mother says that the swelling seemed then about the same size as it was on the first visit to the hospital (about 4½ × 4½ inches; December 4), and from the time she first noticed it she thinks that it "throbbed." On examination the swelling is round and soft, and the skin quite free over it. It is placed over or a little anterior to the right lateral fontanelle. On deep pressure there can be made out some deficiency in the skull, the edge being very irregular and everted especially anteriorly; the deficiency seems to be chiefly in the posterior part of the parietal bone. The skull as a whole is asymmetrical. There is very marked impulse and increased tension on coughing or straining on pressure when the swelling is lax; no definite arterial pulsation can be made out. Pressure on the swelling does not affect the child at all. No history of fits.

The tumour has not been tapped.

The first thing to determine with regard to this case is whether it is an encephalocele or a meningocele. The diagnosis of the former cannot be made with any certainty because of the doubtful arterial pulsation, and in the definite absence of the latter we must still remain in doubt because it has been proved in other cases, which have been con-

sidered certain meningoceles, that the tumours have had cerebral contents. Should brain substance be present the cyst communicates probably with the lateral ventricle. If it be considered to be a meningocele only, which is the more likely, then we must consider if it be congenital or traumatic. Congenital meningoceles through the lateral fontanelle are very rare, and in this position the tumour is perhaps as, or more likely to be an encephalocele than meningocele.

Suppose we were to assume that it is traumatic in origin, we have the factors of a difficult labour, possibly due to contracted pelvis, terminated by forceps. It is not extravagant to imagine that the indentation produced by the latter may be attended by a fracture or separation of bones associated with a rupture of the closely underlying dura mater.

Note.—Since this case was exhibited the tumour has been tapped. Several ounces of clear fluid were withdrawn, so that the sac was quite lax. An irregular hole about the size of a shilling was felt in the posterior inferior part of the parietal bone, but the bone around the opening was not everted, and was quite smooth. At the margin of the swelling was a large rim of new bone deposited from the raised periosteum. There was no evidence of any cerebral contents in the sac. The child experienced no ill results from the tapping, and in a few days the swelling was as large as before.

XIII.—*Case of Cystic Hygroma in an Adult.* By Rickman J. Godlee, M.S. *Exhibited February* 8, 1895.

THE patient, who is a shorthand writer 41 years of age, was born with his present trouble. He remembers that when he was a boy there was a large swelling of the neck and side of the face and under the tongue, principally upon the right side, which used to swell up and turn blue-black with cold. His face was much distorted. He thinks that it gradually diminished in size; but it was liable to attacks of acute inflammation, during which his temperature rose

and he was very ill, and suffocation threatened from the manner in which the tongue was forced upwards and backwards by the swelling of the floor of the mouth. It was during one of these attacks that I first saw him in May, 1890; his temperature at that time being 103° to 104°, and his condition really alarming.

It subsided, however, and as the cysts in the floor of the mouth gave him the most trouble, and seemed always to be the starting-point of the acute attacks of inflammation, I determined to remove some of them. At that time there was a large swelling occupying the right cheek, floor of the mouth, and submaxillary region, and extending part of the way down the neck and slightly to the opposite side of the neck. Part of the mass felt as if it were solid, but the greater part was obviously composed of larger and smaller cysts, some of the largest of which projected into the floor of the mouth. The tongue was, and is still, covered on its upper surface with great papillary and nævoid masses, and countless minute cysts appear on the under surface, so that it is folded over like a huge epiglottis. Some of the lumps appeared to be enlarged lymphatic glands.

In November, 1890, I removed, through the mouth, several of the larger cysts, with less difficulty than I had anticipated, and was pleased and surprised to find that no serious inflammation followed the operation. I had been quite prepared for such an event.

Since the operation he has been much more comfortable, and the deformity has been much less obvious to the casual observer. He has quite lost the fits of *bleeding* to which he used to be subject in hot weather, and has had no attack of severe inflammation. Sometimes he has had warnings, when he has retired to the house and they have always hitherto come to nothing.

He came to me in November, 1894, complaining of a swelling below the zygoma, which extended over the ramus of the jaw and for some distance behind it. The swelling was hard and tender, and the skin over it was somewhat œdematous. It interfered with the opening of the mouth. I am not prepared to say what tissues were involved, but I should think it was in or closely attached to the masseter. I gave him some iodide of potassium in five-grain doses, and afterwards five minims of Liq. Sodæ Arseniatis three times a day, and told him to rub some Ung. Pot. Iodid. over the swelling.

Under this treatment the lump has got less tender and

smaller, but there is no appreciable difference in the degree to which he can open his mouth; so I assume that the swelling is caused by fresh chronic inflammation in some of the lymphatic tissue of which the mass is composed.

I think there can be no doubt that this is an example of cystic hygroma, combined, no doubt, with a certain amount of nævoid structure; and as it is the first case that I have met with in an adult, I have thought it worthy of showing at the Clinical Society. What becomes of the numerous children one sees with this affection I do not think is known. I have operated on several of them, and when the tumour has occurred in other parts of the body have occasionally found, as is, I suppose, always the case when they occur in the neck, that the disease extends so deeply—say through an intercostal space or through the whole thickness of the abdominal wall—that it was impossible to extirpate it completely. One such case operated on when the patient was an infant returned some years afterwards with a recurrence, which I again removed, and have not heard of him since. No doubt some of the children die of the acute inflammation to which they are all liable; but it would be of great interest to know whether cystic hygroma, which I suppose we all agree is a lymphatic nævus, has a tendency, like that shown by so many vascular nævi, to disappear spontaneously as the children grow older.

XIV.—*A case of* Incomplete *Graves's Disease associated with Nasal Polypi.* By Scanes Spicer, M.D. *Exhibited November* 9, 1894.

EMILY B., a neurotic, excitable girl, æt. 17, by trade a bootmaker's apprentice, was sent to the Throat Department of St. Mary's Hospital by Dr. Doidge, of Haverhill, October 23, 1894, chiefly for treatment of the growths in the nose.

On admission she was observed to have some stiffness or impaired mobility of eyeballs, and the width of sclerotic showing between cornea and lids was excessive, causing an appearance of undue prominence of eyes. The thyroid gland

was a well-defined swelling the size of a Tangerine orange on each side, but larger on the right than on the left; the isthmus was not conspicuously enlarged; the tumour was soft, elastic, pulsating, and conveyed a distinct thrill to the hand. On extension of arms and hands a fine fibrillary tremor was well marked. Her respiration rate was 24, pulse rate 140, temp. 98·2°. Both nasal passages were completely blocked with polypi which presented at nostrils, and the "frog-face" deformity was marked.

History.—Her mother discovered the neck swelling and nose blockage at the same time, now some three years ago, and patient was aware of their co-existence before she received any nasal treatment. The obstruction and goitre have gradually increased up to the time of admission. An attempt was made in the country to clear the polypi, which gave her some temporary relief. She has never been conscious of excessive palpitation of heart, nor has her hair fallen out.

Treatment.—At St. Mary's Hospital, up to the date of report, she has had four different sittings for the removal of polypi under cocaine with the cold wire snare. A great many have been removed, and the bleeding has been freer than usual. The nasal passages are gradually becoming pervious. *Pari passu* with this improvement the thyroidal swelling is diminishing and is less defined; the pulsation is less; the slight exophthalmos and tremors have disappeared. The tachycardia is less: usual rate in bed 120, in the chair before operation 136—144, under excitement of operation 196, after operation 160. On the last two occasions on which she has been examined after the moderate exertion of walking from ward to operating chair the rate has been 120 and 132, as against 136—144 after similar exertion before.

Remarks.—Although the thyroidal enlargement, the tachycardia, the tremor, and nervous excitability are well marked in this case, I have entitled it *incomplete* Graves's disease from the subordinate character of one of its leading symptoms —the exophthalmos. The case is shown to afford objective evidence of the facts (1) that Graves's disease and intra-nasal disease may coexist previously to and without the excitation of any intra-nasal operation; (2) that relief of the nasal disease may be followed by amelioration of the Graves's disease.

With reference to the first fact, this case differs from that reported in the *Clin. Soc. Trans.*, vol. xxii, p. 233, and in

which certain symptoms of Graves's disease appeared after intra-nasal operation. Although this latter case was described as an incomplete form of Graves's disease by such distinguished clinicians as Dr. Felix Semon and Mr. Nettleship, I venture to doubt if sufficient proof was adduced to place it in the category of Graves's disease. In Dr. Semon's case, in addition to the absence of thyroidal swelling (not enough in itself to exclude it if sufficient positive signs pointed in the other direction), there was an absence of that general nervous excitability, of cardiac excitability, of *marked* tachycardia, and of tremor. Moreover the exophthalmos was unilateral, and has remained so. Nor has the subsequent development of that case (*Proc. Laryng. Soc. of Lond.*, November 8, 1893), in spite of some permanent increase in the pulse rate and the supervention of alopecia, yet placed it decisively to my judgment in the category of Graves's disease —from which in general aspect and character it differs. That the intra-nasal operations in Dr. Semon's case led to some pathological condition at the back of the orbit and so caused the ocular symptoms, I think extremely probable. His case would then fall into line with the cases referred to by Mr. Cresswell Baber and Mr. Charsley (*Proc. Laryng. Soc.*, loc. cit.), and which developed ocular symptoms yet not Graves's disease and which recovered completely. I have, at least *once*, been told by a patient, on whom I had operated intra-nasally, that the eyeball felt stiff and rigid; this sensation soon passed off. I was not at the time aware of the significance of the symptom, and made no record of it. What the nature of this influence extending from the area of intra-nasal operation into the orbit—whether congestion, inflammation, or œdema of cellular tissue or of muscles or nerves extending by contiguity from the nose, the ethmoidal or frontal accessory sinuses; or vaso-motor spasm or paresis; or thrombosis; or emphysema by opening up orbital connective tissue to ethmoidal sinuses; or muscular spasm followed by contracture or nerve paresis, or what not, is at present a matter of speculation only. That such ocular symptoms as are seen in Dr. Semon's case may be persistent is indeed an untoward matter, but things would be much worse still if it were proved that such a serious disease as exophthalmic goitre had in any case resulted directly from intra-nasal operations.

And here follows the lesson of my case, that even supposing Graves's disease did appear after intra-nasal operation for nasal disease, it must not necessarily be assumed

that the *post hoc* is *propter hoc*, for in the present case we see the association of nasal disease and exophthalmic goitre existing even before the nose was examined.

My case agrees further with those described by Hack and B. Fränkel, and referred to in the latter's interesting and suggestive paper, in showing marked improvement of the Graves's disease symptoms *pari passu* with the restoration of the functional efficiency of the nasal channels. Reviewing the evidence afforded by these latter cases, the connection between intra-nasal disease and exophthalmic goitre appears much more than a coincidence—the most plausible explanation yet given referring it in such cases to some irritation of the intra-nasal terminal sympathetic fibres.

XV.—*A case of Mycosis Fungoides.* By F. DE HAVILLAND HALL, M.D. *Exhibited March* 22, 1895.

R. W., æt. 52, a coal merchant.

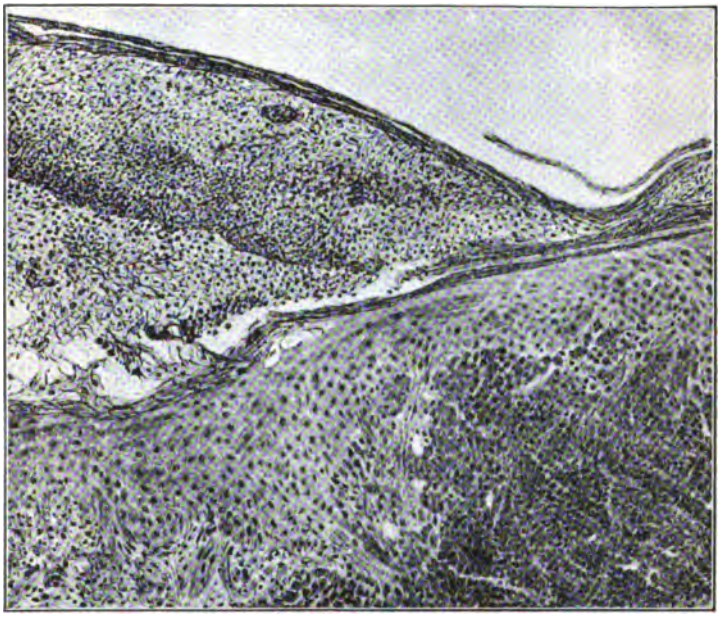
The patient has always enjoyed good health with the exception of an attack of sciatica twenty years ago. Since then he has often had pains in his arms and legs, but he has not been prevented from working. To alleviate the pains he rubbed acetic acid freely over the affected parts for the last ten years. About two and half years ago an affection of the skin began in the places where he had been rubbing the acetic acid. A rash appeared on the outer side of both arms, extending three inches above and three below the elbows. After a time the rash spread to the thighs and trunk.

In September, 1893, a swelling as large as a goose's egg appeared on the right hip, and shortly afterwards another came on the right elbow. During the latter part of 1893 similar swellings appeared on the thighs and then all over the body. The patient states that the tumours lasted from six to nine months each, and then disappeared gradually, some breaking down with the formation of a thin yellowish discharge followed by a scab, and others disappeared without undergoing these changes. The patient was able to continue at work until June, 1894, when the tumours appeared on the

feet, head, and face; since October he has suffered from sore throat.

All over the body are numerous tumours varying in size from a pea to a mass 1½ by 2 inches. Some are quite flat, others are somewhat pedunculated; some are smooth and covered by altered skin, others are ulcerated. The remains of old tumours may be recognised by pigmented and scarred patches on the skin. On the neck there is a papular eruption with some induration of the skin; this is irritable, and itches at times. The patient states that this is the kind of eruption which preceded the formation of the tumours. The glands in the axillæ are slightly enlarged, and there is considerable enlargement of the inguinal glands, especially on the right side.

FIG. 27.



To show the commencing ulceration.

In the pharynx a small elevated mass may be seen on either lateral wall, and a smaller one on the posterior wall. The epiglottis is swollen and superficially ulcerated. On the

left arytænoid cartilage there is an irregular mass about the size of a hazel-nut, the surface of which is ulcerated.

The thoracic and abdominal viscera are apparently healthy, and the patient's general health has not suffered. A tumour has been removed for microscopic examination. Further details are wanting as the patient has been under observation only two days.

The tumour removed was a flattened sphere having a diameter of $\frac{3}{4}$ inch and a depth of $\frac{3}{8}$. There was commencing ulceration of the surface. On section it was found to be a

FIG. 28.

From about middle of section, showing the stroma and cells. × 300.

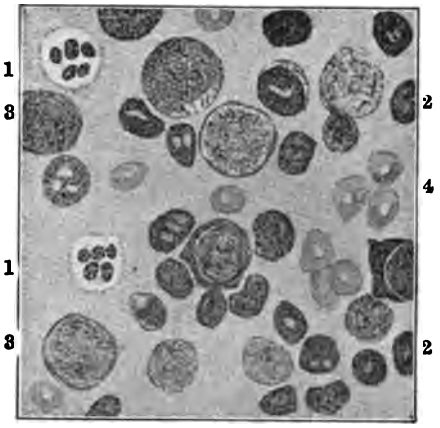
firm though elastic white glistening neoplasm of homogeneous consistence and myxomatous appearance.

Microscopical examination made by Dr. Hebb showed that the dermis was the seat of a lymphomatous growth, *i. e.* there is a reticulum of fine connective tissue the meshes of which are filled with small round cells of uniform size. The nucleus of the cells is large and round, and the cytoplasm relatively very small. Towards the base of the tumour the connective tissue is coarser than in the parts above, and in this there are many vessels containing red corpuscles. There are no degeneration areas in this particular tumour-growth, and the appearances are practically uniform throughout (Figs. 27 and 28).

The surface of the tumour is just beginning to break down, and the sections show that the ulceration process starts as a vesiculation, *i. e.* in the epidermis.

Smear preparations of the freshly expressed juice of the tumour present appearances remarkably like those seen in leukæmic blood. There is this difference, however: in the

FIG. 29.



Smear preparation of piece of mycosis tumour. 1. Polynuclear leucocytes. 2. Lymphocytes. 3. Mononuclear "leucocytes." 4. Red corpuscles. $\frac{1}{8}$ Apochromatic, oc. 10 (Powell and Lealand). × 600 circa.

mycosis juice the number of lymphocytes is much greater, and the number of polynuclear leucocytes much less than in the blood of leukæmia. In both the large mononuclear "leucocyte" is a great feature. Blood from the finger of the mycosis patient merely exhibited some excess of white corpuscles, but in other respects was not particularly noteworthy.

The tumour was fixed and stained by various methods, the results being much alike. There was no evidence of any parasite, vegetable or animal, nor any appearances such as those described by Wernicke in the *Centralbl. f. Bakteriol. u. Parasitenk.*, vol. xii, p. 859 (fig. 1).

XVI.—*Fibrous Nodules of Doubtful Nature.* By R. J. Godlee, M.S. *Exhibited November* 9, 1894.

W. S., æt. 27, a labourer. There was no history of previous illnesses, except an attack of gonorrhœa eight years ago. The question of syphilis, gout, and rheumatism was carefully investigated, and there was no history to be obtained of any of these complaints either in the patient or his family. He was, however, marked by scars of glandular suppuration on the right side of the neck and of some pustular eruption in the occipital region. There was also the scar of a strumous ulcer on the cornea. A few glands could be felt beneath the upper part of the right sterno-mastoid muscle.

His present illness began with a swelling over the bridge of the nose in April, 1894, which was followed a month later by swelling of both eyelids. Afterwards small lumps appeared in different parts of the body, viz. the forehead, hands, back, &c. These swellings caused no pain nor sensation of any kind, and they remained, when once developed, apparently unaltered in size. He was treated at University College Hospital for some time; then he went to the Highgate Infirmary, where he was given iodide of potassium in large doses.

He came under my notice in September, 1894. He was a healthy-looking man, but presented a peculiar appearance owing to the swelling of his nose and eyelids, which looked as if they were œdematous. On pinching the eyelids, the swelling in each felt like a small button under the skin. The swelling itself did not pit like true œdematous tissue. There were a number of small, hard, flattened nodules in, or rather beneath, the skin of the face. The most marked were over the bridge of the nose, placed symmetrically on each side of the middle line; others were in the upper and lower eyelids, on the forehead, and over the lower jaw. All of these were

symmetrical except those on the forehead, one of which was median and the other a little to the left. Similar thickenings were found on the backs of the fingers over the proximal interphalangeal joints, the styloid processes of radii and ulnæ, the internal condyles of the humeri, and the third sacral spine. In the gluteal folds there was marked thickening of the skin, which extended forwards to the inner aspects of the thighs and over the lower parts of the buttocks and round the anus. This thickening had all the appearance of that produced by congestion of lymphatic vessels, and resembled the condition met with in elephantiasis. A somewhat similar condition was present in the skin of the front of the lower part of the forearms. The bursæ over the olecranon processes were much enlarged, and fluctuated.

There was no discharge from the nose, but there was some thickening of the nasal mucous membrane.

The blood was examined on several occasions, and was absolutely normal. There were at this time no hæmorrhages of any description; but since the patient was shown at the Society there have been some slight attacks of hæmoptysis, to account for which no physical signs are discoverable; and as none of the blood has been brought for examination, and there is ordinarily no expectoration, it has not been possible to examine the sputum for the presence of tubercle bacilli.

The spleen apparently was not enlarged.

The temperature whilst he was under observation was usually normal, though it occasionally reached 99·2°.

The patient felt weak. He said he had lost flesh, and did not feel equal to his work. He flushed and perspired freely on exertion.

One of the nodules was removed for microscopical examination. I did not see the specimen myself, as this was done before he came under my observation; but I am informed that it consisted only of fibrous tissue, and certainly not of adenoid tissue.

The thickenings did not present the appearance of rheumatic nodules, nor indeed did they resemble any growths with which I am acquainted.

On the suggestion that the patient might be the subject of an early and unusual form of Hodgkin's disease, he was given arsenic first in the form of Donovan's solution, of which he took alternately ɱxv three times a day, and afterwards he took Liq. Sodæ Arseniatis in the same dose; he could not take larger quantities.

Beyond the almost total disappearance of the enlarged bursæ and the diminution in size of some of the nodules, no change occurred in the patient's condition. He was sent to the seaside, and while there he had the hæmoptysis mentioned above.

He has now, I believe, returned to his work, very much in the same condition as on admission. He has been seen by many, but up to the present time no satisfactory explanation has been given of this very unusual condition of things.

XVII.—*A case of Scleroderma.* By W. D. Herringham, M.D. *Exhibited November* 9, 1894.

William H., æt. 39, seaman R.N., subsequently pearl diver. Diving altogether for sixteen years.

Three years ago he noticed skin on lower part of abdomen getting thicker and harder, and that he did not sweat over it. About six months later the chest and the hands became involved, and the induration spread up to the shoulders. About the same time the legs were affected. About two years ago he noticed affection of face, and that his hair was coming out. A year and a half ago the teeth became tender and felt loose, and since this time he has been unable to protrude his tongue. About the same time he began to be drawn into a stooping position.

During the whole time he has had no pain. There was never œdema of any part at any time, except once in the arm, apparently from an inflammatory condition.

Previous history.—Always good health; no syphilis.

Typical tight-drawn skin, with pinched features, and an appearance of small scars in parts; the hair of the head has grown very scanty; the eyebrows and eyelashes have gone, and the moustache, which was once thick and heavy, is very thin. In axillæ and on pubes no hair. Teeth very loose, gums retracted. Tongue cannot be protruded, apparently from contraction of the mucous folds at the tip, and the surface is smooth and glazed except over the central third. The hands are very deformed from contraction of the fingers at the first phalangeal joints; their skin is smooth and hard, except over the same joints, where it is horny and scaly and marked with

dilated veins. On the right index the skin over the joint was at one time ulcerated. The remaining joints are rigid. There is no shortening of the bones. The skin over the back and sides of the trunk is natural; that over the front of the chest is hard and thick. That over the neck is remarkable; it is marked by transverse ridges running not in the line of any natural folds, but horizontally. Apparently the skin is thickened in the ridges and atrophied in the furrows between. Dilated venules are seen everywhere Power of sensation is not diminished; he thinks, if anything, it is slightly increased. There is a slight amount of albumen in the urine. Other organs healthy.

He was treated for nearly two years at Brisbane, principally with hot baths and inunction of cod-liver oil and lanolin; and for two months at Greenwich with pilocarpine. He was admitted into St. Bartholomew's Hospital under Dr. Hensley (by whose permission he was exhibited) October 13, 1894, and discharged January 25, 1895. He was treated externally with lanolin and alkaline baths, internally, at first with arsenic, and afterwards with thyroid extract and cod-liver oil. There was very slight improvement in the condition during this time, but the skin of the abdomen is softer than it was some time ago.

XVIII.—*A case of Eczema following Typhoid Fever.* By W. Hale White, M.D. *Exhibited November* 9, 1894.

A BOY æt. 10 was admitted into Guy's Hospital, August 27, 1894. He had symptoms of typhoid fever, and he was probably at about the twelfth day of his illness. Spots were observed the day after admission. There was nothing noteworthy about the case, which was moderately severe. The temperature remained below normal from September 14 to September 22, when it rose, and then remained about 100° till October 1. This period of pyrexia was regarded as a relapse, although it was not accompanied by spots. On September 27 a few vesicles appeared on the left upper lip between it and the ala of the nose. These ran the course of an ordinary

eczema, spreading over the face so that by October 8 the disease had spread right round the mouth, and covered an area extending for a distance of one inch and a half from the mouth in all directions. By the next day there was still further spread, so that it reached the neck, the eyebrows, and had extended on to the ears. There was a well-defined edge, the surface was dusky, red, and covered with vesicles and crusts; there was a good deal of serous exudation and much itching.

By October 13 it had spread still further, both eyes were completely closed and the eyelids were very red and œdematous; there was much discharge from the left eye. From this time he gradually began to improve, so that by October 29 there were only a few crusts left on the nose and ears, and they soon disappeared. The treatment consisted of a mask for the face which was smeared with an ointment consisting of 16 grains of yellow oxide of mercury to an ounce of soft paraffin.

XIX.—*A case of General Psoriasis cured by Thyroidin.* By Albert Wilson, M.D. *Exhibited February* 8, 1895.

A WOMAN affected on elbows and knees for years; last August it commenced to spread all over the body, even on the scalp, causing all the hair to come off.

In October thyroid treatment was commenced, and the knees and elbows cleared first. By the end of the year the skin was clear and supple and hair commenced to grow. Thyroidin was stopped in January. There was no depression while taking the 15 grs. of thyroidin (Allen & Hanburys) three times a day.

XX.—*The Treatment of Wounds and Ulcers by Oxygen.*

By GEORGE STOKER. *Exhibited February* 8, 1895.

CASE 1.—Barbara T., æt. 56, married, no children, family history good. The patient has suffered from chronic ulcer of the left leg for more than eight years. She first suffered from pustules on the skin; for this she was treated in the out-patient department of St. Bartholomew's Hospital for thirteen months. After that several small ulcers formed, and for these she was six years ago treated as an in-patient at the London Hospital for seven weeks. There was little or no improvement, and she was sent to the out-patient department, where she was treated for nearly one year, when

FIG. 30.

she was again admitted as an in-patient, and remained in hospital for another period of seven weeks. Again no improvement took place, and once more she was treated as an out-patient for about twelve months.

About four years ago these small ulcers coalesced and formed two ulcers the size of five-shilling pieces, one on either side of the shin with a narrow piece of healthy skin between them. For these ulcers she was again treated as

an in-patient for seven weeks, but they did not heal. Since then, up to the time she came under my care, she attended the out-patient department once a week, but in spite of

FIG. 31.

various forms of treatment the ulcers continued to increase in size, though they diminished in depth.

When I first saw her there was a very extensive ulcer, almost annular (Figs. 30 and 31); there was only about 1½ inches of skin remaining on the posterior aspect of the leg between the outer and the inner borders. The ulcer was dry, hard, and cartilaginous-looking, and of a yellowish colour; the edges were raised and tough, and several warty growths appeared on the surface of the ulcer. The measurements were as follows:

Circumference of leg	10¼	inches.
Length of ulcer on outside of leg . .	5⅝	,,
,, ,, on inside of leg . .	4	,,
,, ,, along shin . . .	3½	,,

She had constant pain, at times very severe. There was very little discharge from the ulcer at any time.

On January 4, 1895, the leg was placed in an oxygen box, and treated by exposure to equal parts of oxygen and purified air.

On January 5, when asked how she had slept, her

answer was, "I have had the first night free from pain for eight years."

Growth of hair similar to that seen in other cases appeared on the leg between the knee and the ulcer. The ulcer continued to heal rapidly, and on the 1st of March the measurements were as follows:

Circumference of leg	5½ inches.
Length of ulcer on outside of leg . .	2 „
„ „ on inside of leg . .	1½ „
„ „ along shin . . .	⅛ inch.

At the end of the eleventh week they are as follows:

Circumference of leg	3⅞ inches.
Length of ulcer on outside of leg . .	2 „
„ „ on inside of leg . .	1¼ „
„ „ along shin . .	quite healed up.

The remarkably rapid progress of this case may be seen by comparing the photographs taken before and during the treatment with the ulcer as it now is (Figs. 32 and 33).

FIG. 32.

The new skin and tissue is not cicatricial, but resembles the normal skin and tissue of the leg.

The discharge from the wound was stained and examined microscopically for micro-organisms before the treatment was begun. It was found to be full of cocci and bacilli, but

FIG. 33.

further examinations showed that after the tenth day these micro-organisms had practically disappeared.

CASE 2.—Thomas George L., æt. 54 years, family history good. On the 1st of last November he bruised the base of the nail of the middle finger of the right hand. The finger swelled up and got very painful. Three days later he put his hand into a drain to unstop it, and the wound on his finger got poisoned. On the 7th of November he went to hospital. The hand was then greatly swollen, and two abscesses had formed,—one on the back of the hand, between the base of second and ring fingers, and one on the back of the wrist-joint: these abscesses were opened on November 16. After that he attended the hospital three times weekly, and he dressed his wounds twice daily with carbolic lotion. When I saw him first on December 11 his hand and fingers were greatly swollen; at the base of the middle finger there was a rough, ragged looking ulcer about the size of a half-crown piece; the edges were red, swollen, and inflamed; the ulcer was deeply excavated, extending down to the palm of the hand. There was another ulcer on the back of the wrist-joint about the size of a shilling, and presenting the same characters as the one just described; it was three quarters of an inch in depth. There was a little ulceration at base of nail of right middle finger. The hand was placed in an oxygen box

and treated with equal parts of oxygen and purified air, and the wounds were washed twice daily with boiled water.

The ulceration on finger healed in twenty-four hours, the ulcer on wrist in two weeks, and the ulcer at base of middle and ring fingers in three weeks. In addition to this rapid healing of ulcers that previously showed little inclination to heal, there were two very important facts to be noted in this case.

1. The disappearance of micro-organisms from the discharge a few days after the treatment was commenced.

2. The rapid growth of nails and hair on the hand. After it had been exposed to oxygen for about ten days the back of the hand became covered with brown hair.

In view of the remarkable growth of hair as seen in Case 2, I determined to try oxygen for the treatment of baldness.

Case 3.—Jessie M., æt. 8 years, was sent to me by Dr. Abraham. Her history is as follows:—In August, 1894, her hair came off in patches, and for this the hair was cut short and ointment applied. In September her head was shaved and various applications made; amongst others blistering fluid was applied, but the hair did not grow. For three weeks previous to coming under my care she had one tabloid of thyroid extract daily. When I first saw her the only evidences of hair I could find on her head were—

1. A slight downy growth on the left occiput.
2. A few straggling hairs on the right parietal region.
3. A few straggling hairs on her left eyebrow.

These have all since disappeared during the first few days of the treatment. She was fitted with an india-rubber cap, which is stretched round the head, and further kept in its place with a piece of bandage: this cap was filled with pure oxygen morning and night. On the tenth day of treatment a growth of fine downy hair was perceptible all over the head, and now at the end of the sixth week this hair has become stronger and longer, being in some places 1½ inches long.

XXI.—*A case of Recurring Parotitis with Xerostomia.* By W. H. Battle. *Exhibited February* 8, 1895.

THE patient, a married woman æt. 54, was admitted into the Royal Free Hospital, under my care, on November 11, 1894, complaining of swelling of glands of the face.

Two years ago she first noticed a swelling the size of a nut immediately in front of each ear. It was hard and movable, not painful, and the skin was not red. She had not been well for three years before, since the menopause, but thought the swellings came in consequence of the extraction of some stumps twelve months earlier.

The dryness of mouth had been noticed for three years before the swellings appeared.

These swellings gradually increased in size, and in three months had reached the size of hens' eggs; since then they had alternately increased and diminished in size, being "up for a month and down for a month." These variations are symmetrical. When large they are painful, and she suffers from pain in the top of the head, which is tender, so that she cannot rest it on the pillow at night. She says that when the swellings begin to diminish in size she becomes very deaf. No medicine has done any good.

In May she had a rash resembling the one present on admission, which lasted three weeks.

As a child was always delicate; at two years of age suffered from scarlatina: at eleven years had a serious illness, with brain symptoms and abscesses about the hips, followed for a time by flexion of the lower limbs.

Catamenia appeared at twenty, then and always painful; ceased suddenly when she was forty-nine. Was married at thirty, had a miscarriage at four months during the first year after marriage. No children. No history of syphilis. Her tonsils were removed in 1880, she had had a sore throat for months before. Some years ago she had an abscess near the rectum. In 1883, disease of the right lung, and kept her room for three months.

Family history.—Father died, aged twenty-three, of phthisis; mother died aged seventy-one of apoplexy. Only one sister, aged sixty, said to be delicate. Her mother is said to have had rheumatic gout.

On admission.—There was a well-marked erythematous

eruption about the face and body, irregularly distributed (she had been taking arsenic before the rash appeared, also in April before the eruption of similar character appeared in May). She is easily agitated, is a teetotaller and has been for the past eleven years, and before that she drank very little.

On both sides of the face there is considerable enlargement of the parotids, although they are, according to the patient, quiet now. The right one, about as large as a walnut, overlaps the condyle of the jaw, extends nearly to the angle, and backwards to the mastoid. It feels firm and lobulated. The left one is similar in character, but rather larger. On both sides the lobule of the ear is lifted and pushed outwards. The skin of the face has a somewhat shrivelled appearance as from previous distension, and is freely movable over the parotids. These swellings are not fixed. On both sides of the neck the lymphatic glands can be felt.

The mouth is very dry and parched, so much so that the patient has some difficulty in eating, and finds it necessary to drink frequently with the mouth full of food. She cannot taste salt, but calls it potato. The mucous membrane of the tongue, like that on the lips and cheeks, is atrophied, very dry and shrivelled. The papillæ of the anterior part of the tongue have disappeared, the mucous glands of the mouth cannot be felt. Secretion can be seen to come from the parotid ducts, but it is small in quantity and looks thickened. Other salivary glands cannot be felt.

Her teeth have all come away and she has to use an artificial set. Appetite is good; not unusually thirsty; suffers from constipation; chest and abdomen apparently normal. Pulse 60; temp. 98°.

Has had a fair amount of worry, and finds the swelling of the parotids becomes worse when worried. She has muscular tremors when agitated, and she readily flushes.

Urine normal, no sugar.

The cause of the deafness of which she complained was apparently an accumulation of wax in the ears. Nothing abnormal could be seen in the nose, although the patient made complaint of excessive secretion and occasional hæmorrhage. A saline mixture was prescribed.

She left hospital on the 22nd November, having had no attack of parotitis; the eruption had gone soon after admission, and she had put on flesh.

In January she came to hospital and said that she continued better than she had been for years; no increased swelling of the glands had been felt for more than two months, and she was much brighter.

In February she came with signs of the commencement of another attack of parotitis.

XXII.—*Case of Excision of Scapula.* By W. Watson Cheyne, F.R.S. *Exhibited November* 9, 1894.

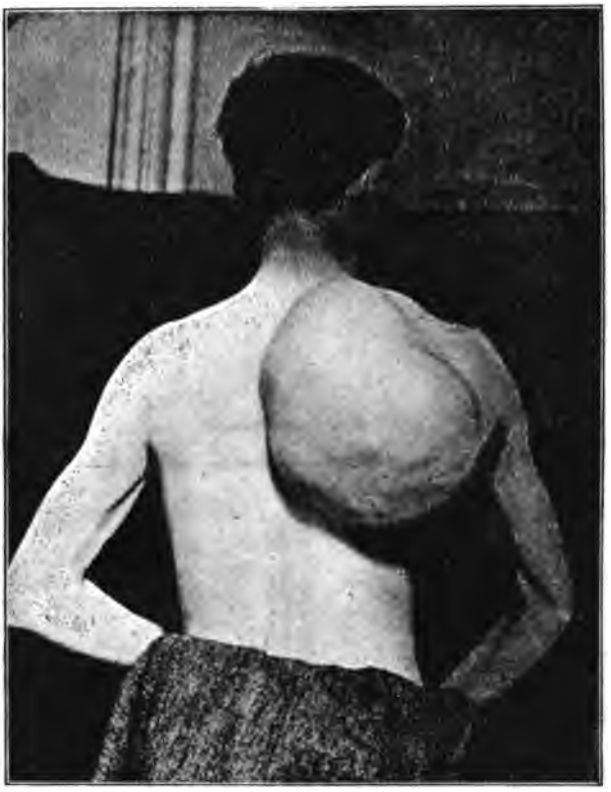
W. B., æt. 50, admitted to King's College Hospital on September 27, 1894. Patient stated that he first noticed a very small, painless swelling on the middle of the dorsal surface of the scapula about seven years ago. This tumour had steadily increased in size and extent since that time, but till a week before admission it had not caused him any pain.

Present condition.—Patient is a small, wiry man, apparently in good health. There is a very large, hard, nodular mass occupying the whole extent of the right scapula with the exception of the acromion process. It overlaps the spinous processes behind. It fills up the axilla, extending as low as the fifth rib in the axillary line and reaching to within an inch of the nipple; it projects forward the pectoral muscles to a marked degree. The arm cannot be brought to the side, but at the closest point forms an angle of 30° with the body (see Figs. 34 and 35).

On October 3 the following operation was performed. In the first instance an incision was made along the lower border of the pectoralis major, opening the axilla freely in front. The axillary vessels and nerves were then searched for, and freed from all connection with the tumour throughout their whole extent. The subscapular artery was then found and tied, and afterwards the pectoralis minor and the other muscles attached to the coracoid process were divided. The patient was then turned over and an incision made along the spine of the scapula right out over the acromion process, and from this two incisions were carried to the lower part of the middle of the tumour, enclosing a considerable portion of redundant skin. These flaps were then dissected off and the scapula rapidly removed, the tip of the acromion, which

appeared to be healthy, being, however, left. The bleeding vessels, which were few in number, having been tied, the detached portion of the deltoid was united to the trapezius, and the muscles around the head of the humerus to the chest

FIG. 34.



wall, the wound was stitched up, a drainage-tube being inserted at the lower angle.

The tumour weighed 10 lbs. 1 oz., and involved the whole of the dorsum and sides of the scapula, but did not project on the under surface. It was an enchondroma.

After-progress.—The shock after the operation was not very great: there was a good deal of bloody oozing. There is nothing remarkable about the after-progress. The wounds healed throughout by first intention, the stitches and drainage-tube being removed on the seventh day, on which day also he

got up for the first time. He left the hospital on October 19, sixteen days after the operation, saying that he felt well, the wounds being soundly healed and there being considerable movement at the shoulder. He was told to continue the use of a sling and not to trouble about movement.

FIG. 35.

When shown at the Society five weeks and two days after the operation, there was considerable forward and backward movement and some power of raising his arm from his side. Attention was specially drawn to the advantage of the anterior incision in all cases of excision of the scapula, enabling the surgeon to detach the axillary vessels and nerves from the growth, to tie the subscapular artery, and to divide the structures attached to the coracoid process, which otherwise is the most troublesome part of the operation.

XXIII.—*Nephrectomy for Malignant Tumour in a patient under two years of age. Read January* 26, 1894. *Child shown in good health two years and four months after operation.* By JOHN D. MALCOLM, C.M.Edin. *Exhibited April* 22, 1895.

THE special interest of this case lay in the long survival of the child after the removal of the right kidney and an oval tumour, 6 inches long, which had been regarded as a malignant adenoma. The operation was performed on November 15, 1892, the child being then under two years of age. When the patient's history was recorded in January, 1894,* Mr. Malcolm had thought that only one other case had been reported in which a child had survived nephrectomy for neoplasm more than a year, and in that case death from recurrence had taken place after eighteen months. But in the debate on the paper Mr. Bland Sutton directed attention to two cases that had been overlooked by the author. Both these cases were operated on by Dr. Abbe, of New York,† the children's ages being respectively two years and fourteen months at the dates of the operations. Mr. Malcolm had been informed by Dr. Abbe that his two cases were alive and well when last seen six weeks before the date of his letter (March 12, 1895). One of them would have survived the operation three years on April 12, 1895, and the other operation was performed five days after that on the patient exhibited.

In Mr. Malcolm's case the tumour consisted of tubules lined by columnar epithelium, and collections of shorter epithelial cells arranged as if to form the lining membrane of a tube, but showing no lumen. Mr. Targett had kindly again examined the tumour in various parts since the case was read to the Society, and he could not find any cells resembling striped muscle cells nor any sarcomatous tissue. As no case had been recorded in which a tumour having this structure had been removed during life, it had been suggested in reporting the case that the peculiarities of the growth might account for the prolonged immunity from recurrence. But in Dr. Abbe's cases the structure of the tumours was different. One of them showed "imperfectly developed tubules similar to,

* *Clin. Soc. Trans.*, vol. xxvii.

† *Annals of Surgery*, Philadelphia, Jan., 1894.

though not identical with, the renal tubules," and "irregular patches and rows of cells." Between these masses of epithelial cells there was "connective tissue so rich in cells of various arrangements that it might well be regarded as sarcomatous." The other tumour contained "a large portion of striated muscular tissue mingled with round and spindle-celled sarcoma." It was evident, therefore, that the microscopic characters of the tumours did not seem to account for the success of the cases; at least no structural peculiarity had been noted as common to the three that had not also been observed in tumours which had rapidly recurred after removal.

The only other suggestion the writer could make in explanation of the result was that it might be due to a very free excision of the parts. In his own case he had very carefully taken away every vestige of the capsule of the tumour and kidney with much of the adjacent fat, and also a mass of fat and enlarged glands surrounding the renal vessels, which were cut and tied close to the aorta and vena cava.

XXIV.—*A case of Tubercular Disease of the Prostate with Fistulæ externally and into the Rectum; treated by Sulphur.* By W. Arbuthnot Lane, M.S. *Exhibited November* 9, 1894.

E. W., æt. 36, had fourteen years ago tubercular disease of both testicles following an injury; this cleared up after prolonged drainage of abscess cavities. A perineal abscess developed thirteen years ago, accompanied by great pain on micturition and discomfort on defæcation. This was laid open about eight months after it first appeared, and urine and pus continued to discharge through the opening. This condition, which was very painful and associated with very considerable general wasting and profuse nocturnal sweats, continued. Seven years ago another mass formed about the scrotum; this broke down, opened externally seven months afterwards, and continued to discharge pus.

Nine months ago a deep-seated pain developed apparently

high up in relation with the front of the bowel, associated with great pain in defæcation and micturition. After some weeks he felt a sudden yielding accompanied with intense pain deep in the part, and followed by an abundant discharge of pus from the anus when his bowels were opened just after. Urine ran freely into the rectum, a small quantity escaping through the urethra and perineum. This continued; he could control the perineal discharge, but not that from the rectum. Other abscesses formed at intervals in the perineum and ischio-rectal fossa.

On June 5, 1894, I laid open a very large irregular branching cavity—or rather a series of cavities—which discharged externally in the perineum and scrotum by many openings, and which extended up into the prostate, and about the membranous urethra. The prostate was apparently much disorganised. Behind the prostate there was an opening into the bowel through which the end of the finger could be passed, and the rectal mucous membrane about the aperture was deeply ulcerated, the margin of the ulcer being thick and raised. The abscesses extended backwards to the right of the anus up along the rectum. The distribution of these abscess cavities was so extensive that it was impossible to expose them fully without doing much harm. The cavities were cleared of their soft caseous contents as thoroughly as possible, and a packing of iodoform gauze was introduced. In a little time his condition improved, but on September 4 he returned in a state which was practically much worse than before the operation. On this occasion nothing was done beyond the periodical injection of sulphur and glycerine into the sinuses. This caused him some discomfort about half an hour after the injection of the sulphur. The swelling in the perineum quickly subsided, the discharge externally diminished rapidly, and the flow of urine from the bowel ceased after about a month of this treatment. He gained over a stone in weight; on September 4 he weighed 8 st. 11 lbs., and now he weighs nearly 10 st. His appearance has completely changed in the last two months. From being miserable, despondent, and wasted, he has become moderately fat, hopeful, and cheerful. The opening in the rectum has closed, and the ulcerated surface has healed. A drop or two of urine escapes occasionally from a small aperture in the perineum, through which glycerine and sulphur are still introduced. Within a month at the most I think he will be quite well and relieved completely of a trouble which has steadily progressed

and resisted treatment for many years, and has made his life a burden.

XXV.—*Traumatic Rupture of the Upper Wall of the Membrano-prostatic Urethra behind the Triangular Ligament, simulating Extra-peritoneal Rupture of the Bladder.* By E. Hurry Fenwick. *Exhibited November* 9, 1894.

I HAPPENED to be operating at the London Hospital at 9 P.M. on May 4, when this patient—a well-built young fellow of twenty years of age—was brought in from Grays with a note from his doctor, Mr. J. R. White, to the effect that the bladder had been ruptured, and that immediate operation was necessary.

At 12.30 the same morning the man had been excavating and using his pickaxe in the stooping posture, when the earth gave way and buried him. He had passed his water shortly before this accident happened. On being extracted he complained of a wish to urinate, but could not pass urine. Mr. White introduced a catheter, and drew off a little blood. When I saw him at 9 P.M. he was in no distress, the belly was neither tympanitic nor unduly tender. Above the pubes as high as the umbilicus and extending to the right was an evident swelling, which was dull on percussion. No urine had been passed. I diagnosed an extra-peritoneal rupture of the bladder for the following reasons:

1. The bladder had been emptied just before the accident.

2. The crushing weight had fallen on the man's back, and the literature of 272 cases (Rivington) has shown that under these two conditions there is often separation of the symphysis and tearing of the bladder low down upon its anterior wall, just above the prostate.

3. The area of dulness above the pubes was an exact reproduction of the condition I had noted in a case of extra-peritoneal rupture which I had the opportunity of dissecting in 1884 (*Pathol. Trans.*, 1886, vol. xxxviii, p. 303).

4. There was no appearance of shock or any peritoneal symptoms pointing to an intra-peritoneal rupture.

He was at once taken to the operating theatre, and a silver catheter passed along the urethra. It appeared to slip easily into the bladder, and I therefore threw a little water along it. The water did not return, and as I could not rotate the instrument at all, I made a small supra-pubic incision, expecting to find the post-pubic space full of urine.

As the deeper layers were approached they were seen to be very dark, and on opening the cavity of Retzius a quantity of blood and black clot rushed out, but no urine was detected. The blood had evidently been kept in the cavity under considerable pressure. After scooping out the clots a large extra-peritoneal cavity was discovered, reaching up towards the umbilicus, and down behind the pubes to the neck of the bladder and laterally. I now noticed that much bright blood kept pouring out from below, as if from some large artery. The patient was placed in the Trendelenberg position, the deep cavity rapidly sponged free, and a strong light from Washington Isaac's search lamp was thrown on to the bladder neck. Each part of what was supposed to be the front aspect of the bladder was carefully examined for the rupture, whence I expected to see the bleeding issuing, but nothing of the kind was discovered. On cleaning away the clot and blood from the bottom of the cavity I saw several large veins had been torn across, and were pouring out a large amount of blood. These veins I found belonged to the plexus of Santorini, and the dorsal vein of the penis as it turns under the pubic arch. Standing upright amidst them and reflecting the electric light from its polished point was the beak of the catheter I had passed, and which I thought had entered the bladder.

The vessels were temporarily clipped with long catch sponge forceps, and a search made for the bladder. Careful palpation of the back wall of the cavity revealed an elastic swelling. It was ovoid and like a semi-full bladder. I dissected down upon it in the middle line, and recognising the vesical muscle, I opened the bladder, and evacuated about six ounces of perfectly clear urine. It was now evident that the urethra had been torn through on its upper wall, in front of the vesical sphincter, either through the substance of the prostate or just in front of that body; that the catheter I had inserted had traversed the deep urethra until it had reached the rupture, through which it had turned up behind the pubes into the cavity of Retzius, and that the bladder, empty at first but subsequently filling slowly with urine, had been thrust backwards and upwards by the increasing

pressure of the wedge-shaped mass of blood which had insinuated itself between it and the pubes. No actual fracture of the pelvis was discovered,—probably the symphysis had gaped.

The further history of the case is uneventful. The deep wound was plugged with iodoform gauze. A soft double tube was placed in the wound which I had made in the front wall of the bladder, brought across the space, and out at the supra-pubic incision in order to keep the pre-vesical wound dry. Next day the gauze plugs were removed and replaced by drainage-tubes. In a week's time a grooved staff was passed into the bladder being guided through the prostatic urethra, and the deep urethra was opened upon it from the perinæum, and a catheter passed into the bladder from below. The supra-pubic drains were then removed, and the entire cavity and wound gradually healed. He is now very dependent on perineal drainage, for directly the catheter is removed the scar tissue in the neighbourhood of the prostate contracts, and he has retention. His subsequent progress will be reported later on.

The case is interesting as exemplifying (1) the close similarity between rupture of the deep urethra behind the triangular ligament, and extra-peritoneal rupture of the bladder; (2) the tearing through and extensive hæmorrhage from the large venous trunks at the apex of the prostate, which is unusual.

I venture to suggest that in all cases of rupture of the bladder in which a doubt exists as to where the actual lesion is situated, the cavity of Retzius should be first explored before the peritoneum is opened. This will do no harm even if no urine is evacuated, and it can be carried out by any practitioner. I mention this because the literature affords abundant evidence of how readily lives might have been saved by a simple incision into the supra-pubic area, for intra-peritoneal ruptures are only twice as frequent as extra-peritoneal (172 : 90—Rivington).

XXVI.—*Reduction of a Traumatic Dislocation of the Hip of five months' duration by the open method.* By W. G. Spencer, M.S. *Exhibited February* 8, 1895.

A BOY, æt. 7, was riding on the limbers of a roller in a pasture-field when he fell off, and the roller passed over his legs. The dislocation was manipulated by a doctor, and stated to have been reduced. An inside splint was applied, which cut into the patient's fork. After six weeks he got up very lame. Five months afterwards there was a typical dislocation of the right hip on the dorsum ilii. The head of the bone slid up to the crest of the ilium when weight was put on the leg. Upon manipulation both before and after the incision the head of the bone travelled easily round as far as the ischium, and there met with an absolutely unyielding ischio-femoral band with the femoral artery in close proximity. It would have been quite easy to have fractured the neck of the femur had more force been applied. Moderate extension was equally ineffectual. A long anterior incision was made, and the acetabulum easily exposed in this way. It was found filled with dense fibrous tissue, which was intimately united with the rectus femoris tendon. The acetabulum was cleared out. It could not have been reached by a posterior incision without resecting the head of the bone. After dividing about two thirds of the gluteal muscles attached to the great trochanter, the head of the bone was drawn down into the socket. But it did not seem to fit well into the cavity except when the leg was slightly abducted and rotated out. The limb has been fixed, since healing, in plaster. Now that the plaster has been removed the limb is normal except for a fibrous rigidity in the hip, with slight abduction and rotation out. He will be encouraged to use his hip freely, and I have no doubt will regain full use of the joint.

The case seems to be one which supports Astley Cooper's theory, and to have occurred from over-*adduction* and driving up. Astley Cooper's theory of the production of a dorsal dislocation has been supported by several recent cases in which the hip has been operated upon. It seems probable that my case would have been, immediately on its occurrence, reducible by extension rather than by manipulation. That some dislocations should occur by over-adduction and driving up does not affect the statement of Bigelow that the majority

occur by *abduction* and rotation of the head round behind the acetabulum. But it is important to note the different manner of production in any case, in order to distinguish those amenable to manipulation from those in which extension is more likely to be successful.

XXVII.—*Two cases of Dorsal Dislocation of the Hip treated by the method described in the* Transactions * *of the Clinical Society.* By W. Arbuthnot Lane, M.S. *Exhibited February* 8, 1895.

PERHAPS I might remind the Fellows of the Society of the principle involved in this operation. It consists in replacing the insecure joint on the dorsum ilii by a secure one beneath the anterior inferior spinous process. The head of the femur is fixed in a cavity cut in and beneath the anterior inferior spinous process of the ilium, and the femoral part of the anterior portion of the capsule is sewn firmly to the fibrous tissues about the anterior inferior spine and to the straight head of the rectus femoris at its origin, it having been cut carefully away from its attachment to the innominate bone so as to have quite enough to make a strong retaining ligament in its new position. The thigh is rotated outwards and fixed in a suitable apparatus with the foot at an angle of about 50° or more with the vertical. By this means the head of the femur is retained in intimate contact with the newly formed excavation, and a minimum strain is exerted upon the sewn ligament.

After some time movements are commenced in the acquired joint, and these soon become very free.

A joint is thus formed which permits of no vertical play of the bones upon one another, and which is very secure, allowing of the ready transmission through it of the superjacent weight of the body. Most important of all is the fact that the new articulation lies in front of the transverse axis around which the pelvis rotates, namely, a line passing through both acetabular cavities; so that

* "Cases illustrating a New Operation for Dorsal Dislocation of the Head of the Femur, and some Points in the Surgery of the Hip-joint" (vol. xxvi, 1893).

instead of there being an ugly, insecure, and often incapacitating lumbar curve with an unpleasant forward projection of the abdomen, the curve of the lumbar region is obliterated owing to the rotation of the pelvis in a direction the

FIG. 36.*

reverse of that present before. Again, the shortening that now exists can be compensated for without exaggerating the deformity, and with the result of tending to obliterate any lateral special curve that was previously present.

Of course it was not suggested to perform this operation in any case in which it was possible to bring the head of the femur into the acetabulum, and to retain it there; but my experience would suggest that this can only be accomplished satisfactorily in a very small proportion of cases of any standing.

This lad (figs. 36 to 39) is the first case on which I operated,

* Figs. 36 to 39 represent the degree of movement permitted by the new joint on the right side.

in September, 1892, so that two years and five months have since elapsed. You will see that the joint is perfectly secure, that it permits of every movement which exists in the normal joint, and that it has undergone no displacement in the interval. The mechanical advantage this child has derived from the operation is most remarkable.

FIG. 37.

As one would naturally expect, if the condition of dorsal displacement is symmetrical, the benefit gained by replacing the very mobile joints behind the transverse axis of pelvic rotation by two secure joints in front of it is not nearly as great as when only one joint is affected; still the improvement in the patient in the former case, both as a locomotive machine and in grace of attitude, repays one very well for the comparatively slight risk which accompanies the operation. Also a much longer period must elapse before the patient with the double dislocation is able to acquire a familiarity

with and a perfect control over the altered mechanical arrangements of his bony and ligamentous framework.

A. W., æt. 8 years, was admitted into Guy's Hospital under my care in March, 1894, with as deforming and incapacitating a condition of double congenital dorsal dislocation as I have seen.

FIG. 38.

By means of a vertical anterior incision, associated with one passing backwards at right angles to it, the loose false joint on the dorsum ilii was exposed, after the glutei muscles had been divided over it.

The anterior portion of the capsule was cut through at its iliac attachment, and the stretched and thickened ligamentum teres was divided. There was no evidence of the presence of the original acetabulum, and although the muscles were divided very freely it was found impossible to retain the head of the femur in a normal relation with the innominate bone when

the leg was brought down beyond an angle of 100° with the horizontal plane.

FIG. 39.

A cavity was then cut in and beneath the anterior inferior spine, the head of the femur was modified in shape, and the operation was completed.

Owing to the fact that there was practically no neck, the head of the bone being set almost directly upon the upper extremity of the shafts, it was necessary, in order to prevent the head slipping away from its new attachment, to fix the thigh in such a position of external rotation that the foot lay in a horizontal plane.

In the following August the opposite leg was operated on in the same way. Some difficulty was experienced in getting the head of the bone down to the level of the anterior inferior spine, and it was necessary to encroach somewhat on the notch above it.

In the joint which was first constructed the patient has acquired a very extensive and complete degree of movement, and the amount of movement on the left side is being steadily increased. Some of the limitation of movement on this side is due to the obstacle which existed to bringing down and retaining the head of the bone at the same level as the right.

The joints are both secure, permitting of no vertical play. The very considerable lordosis, together with the associated very objectionable prominence of the abdomen, are both removed, the lumbar spine being straight and the abdomen natural.

Besides, her walk is very much better than it was before the operation, and is improving steadily. The greatest difficulty experienced by the patient was in overcoming the position of very complete outward rotation of the leg in which the limbs were of necessity fixed at the time of the operation, but this she is succeeding in doing very satisfactorily. This trouble would not arise to the same degree in the ordinary less severe case or in younger children. It is obvious that the earlier the cases are operated on the more perfect will be the results obtained.

I have applied the same principle with the greatest possible mechanical advantage to cases of dorsal dislocation from disease of the hip-joint and of ankylosis of this joint in a position of flexion. In the latter condition the results obtained, especially in young people, are infinitely better than those which result from section of the neck or upper end of the femur.

XXVIII.—*Congenital Dislocation of the Shoulder backwards in an Infant.* By Frederic Eve. *Exhibited February* 8, 1895.

HILDA G., æt. 9 months. The baby was delivered with instruments. The mother stated that on the day after birth the left arm was noticed to be paralysed; movements gradually appeared in the hand and forearm. The left upper extremity was shorter than the right, but not thinner. It

was also colder at first, but subsequently became of the same temperature as the other.

Condition on admission to the Evelina Hospital (January 5, 1895).—The child was well nourished and developed. Left shoulder: movements of the left shoulder-joint were obviously limited, the arm being held stiffly and rigidly across the chest about opposite the nipple. External rotation was very limited, and during extreme rotation outwards the dorsum of the hand still looked forwards and slightly to the left. Internal rotation was increased. The outer surface of the shoulder was slightly flattened. A smooth spherical projection, evidently the head of the humerus, could be felt on the dorsum of the scapula just beneath the junction of the acromion with the spine. The articular surface was directed backwards and to the middle line. There was considerable rigidity of the shoulder-joint, but this disappeared under chloroform, when movements of rotation of the humerus were freely communicated to the head. Measurement from the top of the acromion to the external condyle gave half an inch shortening on the affected side. The circumference of the left shoulder was $\frac{7}{8}$ inch less than that of the right; this peculiarity may probably be referred to want of development of the muscles around the shoulder on the left side. There was practically no difference between the circumference of the two upper arms at the middle. The deltoid, biceps, and triceps muscles on the left side responded readily to the Faradic current, but the biceps less freely than that on the right side. The galvanic current also caused a marked reaction in these muscles.

Remarks.—R. W. Smith* was the first to describe congenital dislocation of the shoulder. One of his cases was a woman æt. 42, a lunatic. The deformity was not noticed until after death. On proceeding to make an autopsy a peculiarity of both shoulder-joints was observed, which was found to be due to displacement of the humerus backwards on the dorsum of the scapula. That the displacement of the humerus was a result of malformation was indicated not only by the condition of the articulation but also by the circumstance that the peculiarity was bilateral. The head of the humerus on each side formed a distinct projection beneath and behind the summit of the acromion. There was

* *An Essay upon the Original or Congenital Luxations of the Upper Extremity of the Humerus*, Dublin, 1839.

no trace of a glenoid cavity in the usual situation, but a well-formed socket surrounded by a capsule existed upon the posterior surface of the neck of the scapula. The head of the humerus was of an oval form, due to deficiency on its anterior surface.

Six cases of subcoracoid congenital dislocation of the humerus have been recorded, four of them by R. W. Smith; and two in which the displacement is described as supra-acromial (*sus-acromiale*). There was a subluxation of the head of the humerus upwards and outwards. "The acromion and coracoid processes were equally pushed backwards and upwards."

It is necessary to consider whether the dislocation existing in this case was due to paralysis, to trauma during parturition, or to a congenital malformation of the joint. In regard to the first, it seems clear that the paralysis which is stated to have existed by the mother was simply a pseudo-paralysis due to loss of mobility of the shoulder-joint. No paralysis exists at present, as shown by reactions to electricity. Further, the condition of the joint in this case has nothing in common with the loose "floating" articulation existing in cases of luxation from paralysis.

Subsequently to showing the case an operation for correcting the displacement was successfully performed. The glenoid cavity was perfectly developed, a fact strongly suggesting that the dislocation resulted from trauma, probably during parturition.

XXIX.—*Two cases illustrating Obliteration of Psoas Abscesses after one washing out and scraping and closure without drainage.* By A. E. Barker. *Exhibited February* 8, 1895.

THESE cases were shown by Mr. Barker to illustrate the advantages of a method of treatment advocated by him about six years ago, which at that time was quite novel, and by many considered heterodox. In both cases extensive tubercular psoas abscesses were opened by incision and flushed and scraped with one of his hollow gouges and then

closed completely by careful stitching *without any drainage at all* after some iodoform emulsion had been introduced into the cavity. In both cases primary union was the result, and there has been no trace of recurrence in either case. In one case five years have elapsed, in the other eight months. In both the scars are sound and soft. In the younger patient there was a bad general state to contend with, as the boy was very tuberculous and at the present moment is suffering from active cervical caries. These two cases are members of a considerable series showing the advantage of the method.

XXX.—*Rotation of Vertebræ without Lateral Curvature.*

By W. H. Battle. *Exhibited February* 8, 1895.

P. W., a girl æt. 10, come to my out-patient department at St. Thomas's Hospital in January, 1894, complaining of pain in the back, which had commenced in June of the previous year, and was supposed to have been due to moving the sash of a window.

She was an anæmic child, who presented nothing very marked at first inspection of the back. More careful examination, however, showed a fulness on the left side of the lumbar spine, while the muscles on the right side were less prominent than natural. This appearance was much exaggerated when the patient stooped, which she did without difficulty or rigidity of the spine. The swelling gave the impression of a rounded column of bony character possessing the normal flexibility of the spine, diminishing in size from below upwards, and uniformly firm. On the right side there was less than the usual resistance on pressure over the muscles. There was no curvature, angular or lateral.

Under treatment with tonics she lost her pain, and the condition of the back has altered so that there is now well-marked curvature to the left in the lumbar region with some compensatory curvature in dorsal region.

XXXI.—*Extreme Displacement of the Heart due to contraction of basic cavity of left lung.* By Arthur T. Davies, M.D. *Exhibited February* 8, 1895.

CAROLINE H., æt. 33, single, came under my care in February, 1894. She complained of cough, which she stated she had always been subject to in the winter. About four years ago she brought up blood.

On examination there was found, in addition to left apical changes, a cavity at the left base, extending up to the mid-scapular region. On seeking for the apex-beat of the heart, it was found deeply situated *anteriorly* to the posterior axillary fold. She remained under my care till March, 1894, and I did not see her again till December, 1894, when she came complaining not of her cough, but of "her heart getting in the way of her arm when she moved it." On re-examination I found that the cavity was apparently smaller, and that the apex-beat was apparently situated close under the angle of the scapula, so that it had been still further drawn towards the spine. In addition, a distinct systolic thrill was felt in the upper part of the axilla, and on auscultation a loud dislocation murmur was found to correspond with it. These are the physical signs at the present time, but lately, in addition, at the end of inspiration with each systole of the heart a distinct clicking noise is audible over the cavity. There appears to be entire subsidence of her lung symptoms, and the patient at the present time complains of some dyspnœa, and also of the feeling above stated with regard to her heart in its relation to her left arm.

There is emphysema of her right lung, and a roughening at the aortic orifice.

XXXII.—*Case of a Second Empyema on the same side, eight years after the first.* By Samuel West, M.D. *Exhibited February* 8, 1895.

William F., æt. 15, came to the out-patients of St. Bartholomew's Hospital complaining of cough and shortness of breath. He looked very ill, almost as if he were phthisical, and his temperature was raised. The physical signs showed dulness all over the left side, which was slightly contracted as compared with the sound side. The heart was displaced to the right, the dulness extending one inch to the right of the sternum. Over the whole of the lower part of the dull side the voice and breath sounds were absent, but at the apex they were exaggerated and there was a good deal of medium-sized crepitation, which was increased on coughing and on taking a deep breath.

The signs pointed to the presence of fluid, a diagnosis which would have been simple if the patient had not given a history of an empyema eight years previously, for which he had been operated on in the Evelina Hospital, the scar of the operation being visible in the posterior axillary line.

The diagnosis lay between phthisis with a thickened pleura, or a second effusion. In favour of phthisis were the signs at the apex and the contraction of the side, which measured one inch less than the sound side. On the other hand, the physical signs were clearly those of an effusion, and the only difficulty was to understand how a second effusion could occur on the side which had previously been laid open for empyema.

The patient was admitted into the hospital and passed under the care of Sir Dyce Duckworth. Great difference of opinion was expressed, and on the whole the balance was in favour of phthisis, of which disease his father died. The patient sweated somewhat at night, was losing flesh, and the temperature rose irregularly at night. After a fortnight's time, the patient having made no improvement, a needle was inserted in the mid-axillary region, but no fluid was found. There was a little expectoration, but examination showed no tubercle bacilli. The patient was then allowed to get up, but his condition became worse, and the dulness increased towards the right as if the heart was being pushed still further over. Another exploratory puncture was now made in the

sixth space in the anterior axillary line, and this time pus was found. The next day the side was opened in the mid-axillary line and twelve ounces of pus removed. The cavity in which the pus was found extended to the pericardium on the right, to the diaphragm below, upwards a considerable distance so that its limits could not be reached by the finger, while backwards it was limited by firm adhesions at the posterior axillary line.

The temperature immediately fell to normal, and did not rise again.

There is nothing further to report except that recovery was rapid and uninterrupted. The lungs re-expanded, and on leaving six weeks later the patient was fat and well, the wound was completely healed, the heart in its normal place, and though the resonance on the affected side was still a little impaired, the air entry was fair.

Cases of this kind are very rare, and it is clear that they must be, for after empyema the whole pleural cavity is usually obliterated, so that a second empyema on the same side can hardly occur, except where the original empyema has been localised. That presumably was the case in this patient, a presumption which was confirmed by the condition of things discovered at the time of the operation, for the empyema cavity was limited posteriorly by adhesions corresponding with the position of the old incision.

XXXIII.—*Extreme Stenosis of Lower Part of Pharynx (syphilitic).* By W. H. Battle. *Exhibited February* 8, 1895.

R. R., æt. 24, was admitted into the Royal Free Hospital under the care of Dr. West in June, 1894, for dropsy, and later came under the care of Dr. Calvert, in whose out-patient department he had previously been for chronic interstitial nephritis. He had suffered from a sore throat for two and a half years, and had been treated for that in the surgical out-patient department under Mr. Battle. This affection had been a most intractable ulceration of the upper part of the pharynx and fauces, which was only slightly influenced by

mercury and large doses of iodide. He had also had gummata and eruptions on the skin.

On June 25 he was pale and suffering from dropsy of the legs and scrotum; the urine contained traces of blood, and gave $\frac{1}{3}$ albumen on boiling. The chest showed nothing abnormal then, but in September there was found to be some emphysema, slight noise in respiration, with complaint of difficulty in swallowing solids.

The condition then was practically the same when readmitted under the care of Mr. Battle on September 24, only there was more contraction in the lower pharynx. The soft palate was high and adherent to the posterior wall of the pharynx, the tonsils were not visible, but an arch of cicatricial tissue, very firm to the touch, closed off the posterior nares from the pharynx, excepting for a small median opening the size of a cedar pencil. There was some

FIG. 40.

Syphilitic stenosis of lower pharynx—drawn by Mr. Leonard Mark.

ulceration of the pharynx with contraction. Respiration was a little difficult, voice hoarse, and the man had a slight cough with muco-purulent expectoration. Examined by the laryngoscope, the first impression given was that he had stenosis of the larynx, for a small opening was visible behind the base of the tongue, with a cicatricial margin as shown in the sketch.

But it was soon evident that this opening was not far enough from the mouth, and that there was no epiglottis. When the finger was introduced the opening was easily felt, and from it there was an extension of mucous membrane to the pharynx behind and on each side, and it was evident that this opening was the only passage from the mouth to the larynx and œsophagus. As it was not larger than a No. 12 catheter, surprise was felt that he could swallow as well as he did and without coughing, also that there was not more obstruction to his breathing. On depressing the tongue the opening could be seen, there being a slight amount of cicatricial tissue forming its front margin and extending on to the tongue. The vocal cords could not be seen.

On September 26 a preliminary tracheotomy was done, and Trendelenberg's tampon introduced. Chloroform was given and well taken. A gag was introduced and the tongue pulled forward. A No. 12 black catheter would not pass through the opening. The strictured part could not be dilated with forceps, so incisions were made on both sides with blunt-pointed scissors. The finger, when introduced, found the pharynx below the fresh opening to be irregularly contracted over a distance of from an inch to half an inch, and some of the edges of contracted tissue required division with a sharp-edged palate knife before the finger could be passed. Further dilatation was effected and a tube passed into the œsophagus. The tracheotomy tube was kept in for sixteen days and an œsophageal tube for five days. Dilatation was carried out daily, after removal of œsophageal tube, by means of the finger and several instruments. He left hospital on October 29 much relieved.

There is a constant tendency to contract, and Mr. Templeton, the house surgeon, continues to dilate the opening at intervals.

XXXIV.—*A specimen of Urine rendered Green by Indigo.* By Archibald E. Garrod, M.D. *Exhibited February* 8, 1895.

THE specimen of urine exhibited was sent up from the country by Mr. Harold Owen. It was quite limpid, acid in reaction, and had a tint resembling that of the green Chartreuse

liqueur. It was passed in the early morning by a healthy child 2½ years of age; a second specimen of a few ounces passed later in the morning had a similar but much less pronounced green colour. At 11 P.M. on the previous night the child was found sucking the blue cloth which formed the border of a fur rug, and its tongue was seen to be stained blue. A specimen of the cloth was sent up for examination, and it was found to be dyed with indigo. Nothing abnormal was noticed in the fæces except that on this and the following day they were rather darker than usual. Chloroform shaken with the urine acquired a pure blue colour, and before the spectroscope showed the absorption band of indigo blue; and the urine itself when examined in a depth of 20 centimetres showed the same band faintly. It was therefore evident that the green colour of the urine was due to the combination of the yellow tint of the urine with the blue of indigo. From the appearance of the urine it might have been supposed that the indigo was in solution, which, however, could not be the case, and the fact that the blue pigment was in reality suspended in an impalpable form, was demonstrated by passing the urine through a filter, when it lost much of its green tint, whilst the filter-paper showed a blue stain.

The specimen is of interest because it shows the presence, as the result of ingestion, of a pigment which is sometimes formed spontaneously in urine; but such spontaneous formation of indigo blue is only seen in alkaline urines, which may have a greenish tint, but exhibit also a blue scum or deposit of microscopic particles, and never present the limpid appearance of the present specimen.

The fact that indigo taken by the mouth profoundly affects the colour of the urine was familiar to those physicians who about the year 1832 administered this substance as a remedy for epilepsy.

In a paper by Roth (*Wissensch. Annalen der gesammten Heilkunde*, 1835, xxxi, p. 23) the urine of patients so treated is described as having a deep purple colour, but some examinations made at that period failed to demonstrate the presence in it of indigo as such. The difference in colour between such urines and the present specimen was doubtless due to differences of dose, for whereas in the present instance the amount absorbed was minute, indigo was administered to epileptics in doses of as much as 6 or 8 drachms in the twenty-four hours.

A similar green coloration of the urine due to the adminis-

tration of a blue drug is observed in cases in which methylene blue is administered.

XXXV.—*A new Instrument for Exploring and Draining Cavities.* By G. C. Garratt, M.B. *Shown March 22, 1895.*

THE instrument is intended for exploring, draining, or aspirating any cavity; it is simple, portable, free from taps and screws, is readily cleaned, and can be boiled in a test-tube. It consists of a cannula and trocar made in three sizes.

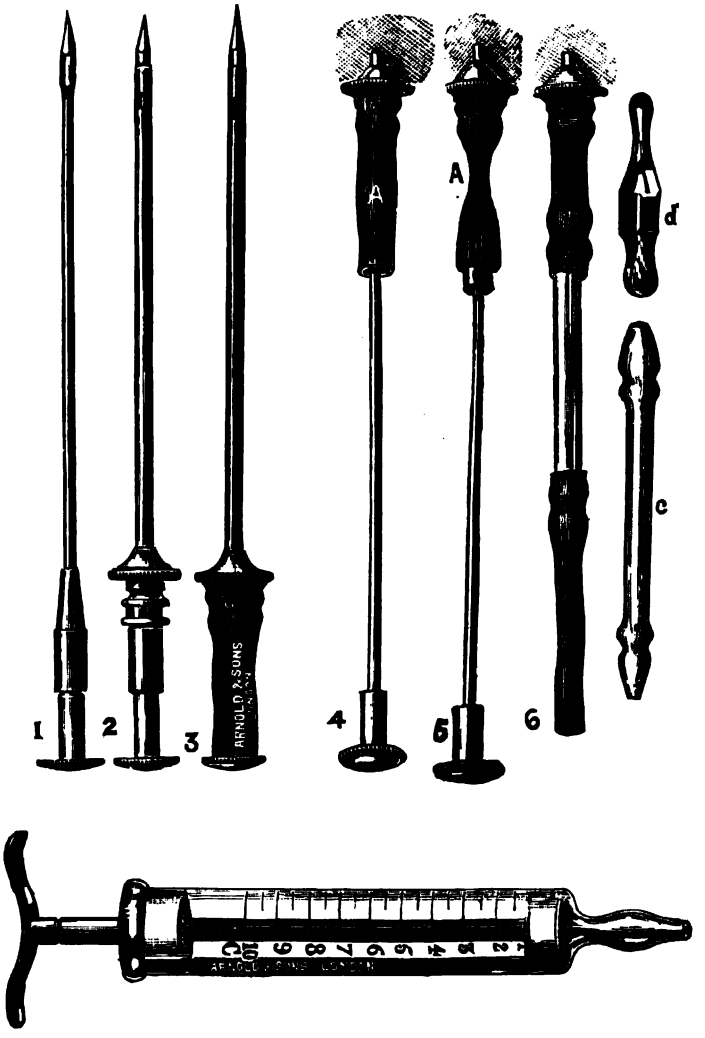
The cannula differs from Potain's in having the mount smooth inside, so that the airtight chamber on the trocar fits directly into it without any intervening tap (Fig. 41, 2).

The trocar (1) has a thick neck ½ inch long; its airtight chamber is a solid metal cylinder, smooth outside and without any concealed washer or packing within; the trocar can be drawn back through it until the point is buried, but no further.

Before use, a piece of rubber tubing 1½ inches long is fitted on to the mount of the cannula, and secured by a thread tied round in the groove provided. When the trocar is put inside, the rubber covers the airtight cylinder, and also the neck of the trocar (3); it is now ready for use. The instrument is thrust into the cavity, and the trocar drawn back into the cylinder (4); in doing this it is well to hold the cylinder at A, lest it be drawn out of the cannula with a jerk. The cylinder is then given a slight twist to insure that it is loose, and is withdrawn through the rubber tube by steady traction on the trocar; while withdrawing it the rubber is pinched with finger and thumb, or clamped with bull-dog forceps to prevent ingress of air (5 and d). The cannula is thus left in the cavity, ready fitted with drain-tube; if exploration is intended a glass syringe is inserted into this tube; if drainage or aspiration is desired, the tube is pinched again and the syringe is replaced by a glass tube (c) previously connected either with a long drain-tube full of water (6) or with a Potain's bottle. Failing the bottle, a Higginson's syringe will replace it for aspiration.

Two bull-dog clips are provided with each set of trocars and cannulæ; each cannula has a blunt rod as well as a trocar to match. The set is sent out in a neat metal box, which can easily be carried in the pocket. To clean the

FIG. 41.



inside of a cannula draw a few threads of soft white darning cotton through it by means of the loop of silver wire provided.

My syringe for exploring is a 10 c.c. glass syringe, the glass having no metal attached to it, so that it can be boiled. The plunger is of asbestos; it can be compressed by an adjustable metal disc, so as to insure always an accurate fit in the barrel. The screw cap in general use in such syringes is replaced by a rubber cork, the piston running in a metal tube through the cork, which has a metal cap overlapping the edges of the glass, and giving a neat finish to the syringe. The cork works better than screw caps, which do not work well on glass, and is much easier to make. The handle of the syringe is very comfortable to hold; every part of the syringe takes to pieces easily and can be boiled.

Attached to a hypodermic needle such as is sent out with the trocars, the syringe will do well for injecting antitoxin.

A pair of test-tubes is sent out with the syringe in which the trocars and cannulæ can be boiled; they are fitted with rubber caps having a slit in the top: these allow the tube to be boiled nearly full without the water bubbling out, the steam escaping through the slit. While boiling make the slit gape by pulling down the cap, afterwards close it by pushing up the cap, and the tube will remain sterile until required.

The syringe and test-tubes are sent out in a polished mahogany box, with 4 feet of drainage-tube.

With a box of trocars in one pocket and the above in another, the practitioner is equipped for the exploring and draining of nearly any pleuritic or ascitic effusion; should he, however, require to aspirate, a Higginson's syringe is usually obtainable. He is also equipped for injecting antitoxin.

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